SCIENCE IN THE SERVICE *of* HEALTH

ELLIOT R. DOWNING

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SCIENCE IN THE SERVICE OF HEALTH

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Longmans' School Science Series

SCIENCE IN THE SERVICE OF HEALTH

BY

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ASSOCIATE PROFESSOR OF THE TEACHING OF SCIENCE
THE UNIVERSITY OF CHICAGO SCHOOL OF EDUCATION

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DOWNING

SCIENCE IN THE SERVICE OF HEALTH

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AUTHOR'S PREFACE

SCIENCE HAS been taught largely for its knowledge value. Instructional material in science is not organized at present primarily to impart skill in scientific thinking nor to develop those emotionalized standards which have motivated much of the research work that has produced science. If pupils do acquire some appreciation of what science has cost, of the heroic endeavors of the great scientists or any realization of the essential elements and safeguards of scientific thinking, it is as an incidental by-product. Yet the history of science and the biographies of the great scientists are replete with incidents of self-sacrificing devotion in the interests of fellow-man, of a stern adherence to truth when falsity would have been profitable, of a firm faith in the moral import of Nature's immutable laws. There are numerous examples of the method of science, opportunities to follow in the footsteps of master thinkers as they have solved scientific problems. Here is material to inspire emulation, to stimulate high endeavor, and generate or strengthen worthy attitudes of mind. If it can be effectively pre-

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sented to impressionable youth it should contribute in no small measure to educational objectives that science instruction has largely neglected.

In this book an attempt has been made to adapt some of this fascinating historical and biographical material to school use. The knowledge content of science has not been neglected. Three principles of science of large importance in solving the problems of the average individual are presented in considerable detail. It is hoped that these will be mastered so they will apply to the constantly recurring problematic life situations. But even more it is hoped that there will be acquired some appreciation of the scientific method and some new ideals, tastes, and mental attitudes.

Formal tests may evaluate the acquisition of knowledge by the pupils or even give some notion of how well he has developed the scientific attitude, though in this last item probably and certainly in his achievement of new emotionalized standards indirect methods of estimating outcomes will probably avail more at present than any attempts at direct measurement.

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SCIENCE IN THE SERVICE OF HEALTH

CHAPTER I

THE PIONEERS

THE INCREASING LENGTH OF LIFE

When our grandfathers were boys, the average person lived to be thirty-five years old. Of course, many people lived to be much older, but many also died when they were younger; so that if the ages of all persons dying in one year in the United States were added together and the sum were divided by the number of such persons, the average was, in round numbers, thirty-five years. Now the average person may expect to live fifty-eight years. The length of life has thus increased greatly within a short time.

Facts like these are found out from the records kept by men interested in life insurance. They must know how much to charge people yearly for their insurance, or they would lose money in the business. The amount of their charge will depend

on the number of years the person is expected to live. Such records are kept, too, by the United States and by other countries in order to know if people are becoming healthier. Evidently they are, and we may all be glad of it. In the time of Shakespeare the average person in England lived to be only twenty-eight years of age and in the old Roman world, the expected length of life was even much less.

THE GERM NATURE OF DISEASE

Likely the most important factor in bringing about this change was the discovery that diseases are usually caused by very tiny living plants and animals. These get into our bodies and destroy our organs or make poisons that kill us. These deadly little germs are very tiny. Suppose one could see the germs of such a disease as typhoid fever. Let him count them at the rate of five a second. That is fast counting. It would take twenty years, working ten hours every day, except Sundays, to count the germs in a space as large as the head of a match. It would take several million such germs placed side by side to cover the butt end of the match.

The germ nature of disease was found out only a short time ago. Men had to discover, first, that there were such tiny living things. Before that could be done, something had to be invented that would enable us to see them. Many people aided

in this discovery. It has usually been true that a great discovery is not made all at once. Many persons each help a little bit, often without knowing they are helping. Then someone puts together

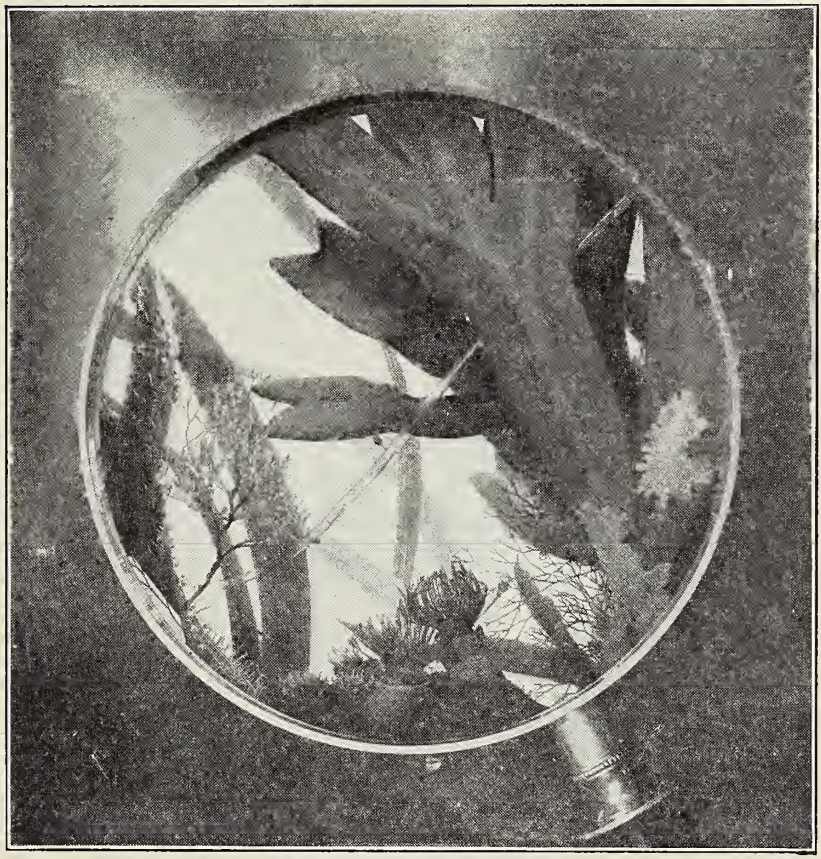


Fig. 1. A reading glass makes things appear larger. Plants and animals of the sea, magnified many times. [Courtesy American Museum of Natural History]

all these parts of the truth and the great discovery is made.

You have probably used a reading glass or lens to make things appear larger (Fig. 1). Such

lenses have been used for thousands of years. They are found in old ruined buildings in Egypt and in still older countries, but it was not until about three hundred years ago that men found out

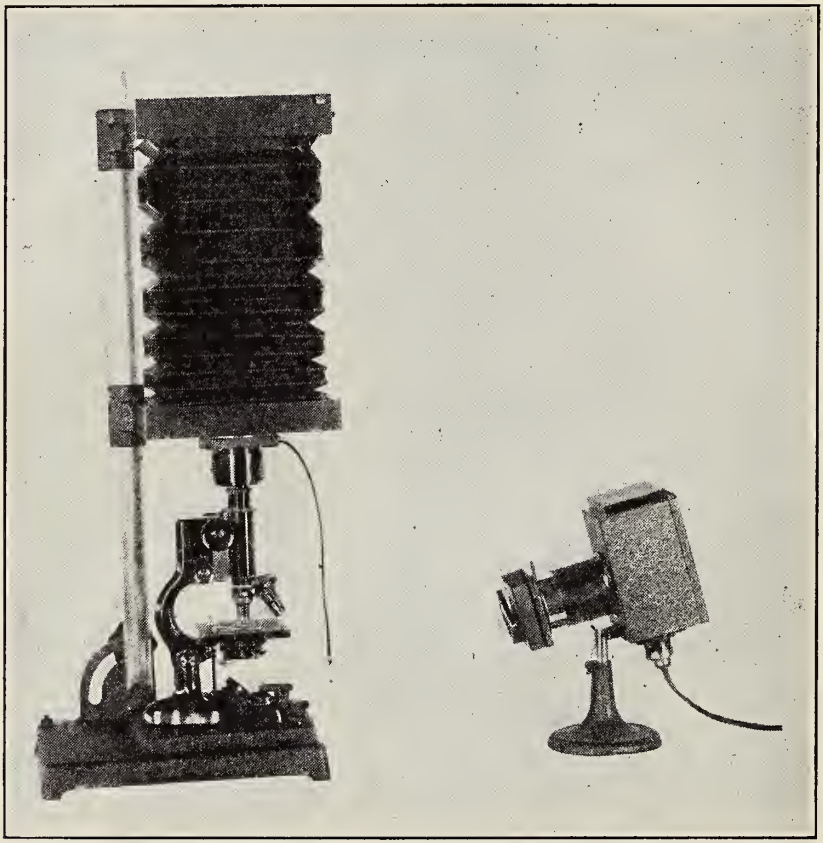


Fig. 2. A microscope set up to take photographs of the magnified object (such as is seen in Fig. 23). [Courtesy Bausch and Lomb]

that by putting two or more such lenses together, things could be made to appear very much larger. This device was called a microscope (Fig. 2). We are not sure just who discovered how to make it, for several people seem to have found it out

about the same time. Probably Zacharias Joannides and his father, spectacle-makers of Holland, are to be credited with the discovery in 1608; Gallileo made a microscope in 1610, without knowing about the instrument made by Joannides, father and son.

DISCOVERY OF BACTERIA

The first man to see the very tiny living things under the microscope was also a Dutchman, Antony Leeuwenhoek (Fig. 3). When he was twenty-one years of age he set up in the dry goods business in the city of Delft, Holland, where he had been born in 1632. He heard that with properly shaped lenses of glass, one could see things much enlarged. He thought that would be fun, so he learned how to grind lenses. The more of the wonderful things he saw with their aid, the more he wanted to see. He became very skillful in shaping lenses and putting them together to make microscopes. It is said that he had some 250 microscopes and some 400 hand magnifiers. His

Fig. 3. Antony Leeuwenhoek [Locy]

microscopes were crude affairs, not much like the ones we use today.

But he was not just a lens-maker. He looked at everything he could lay his hands on. A hair from his head looked like a rope, a cheese mite seemed as big as a horse, a flea's sting was a wonder and the eye of a louse, a marvel. Then one day he looked at a drop of water from the jar that caught the drip from the house roof. And he saw a new world. There were dozens of "wretched beasties" swimming around in it, each so small he could hardly believe there were such tiny living things. They were a thousand times smaller than the eye of a louse. There were many different kinds "swimming about with incredibly thin feet." And now he went hunting for these new sorts of creatures everywhere. He found them by the thousands in the dirty water of the canals. (Fig. 4.) He scraped the food particles from between his teeth and there were new sorts in the scrapings. He saw them in rotting dead clams he fished out of the canals. Some of these tiny living creatures that he saw in the dead clams and in the scrapings from his teeth were bacteria, the

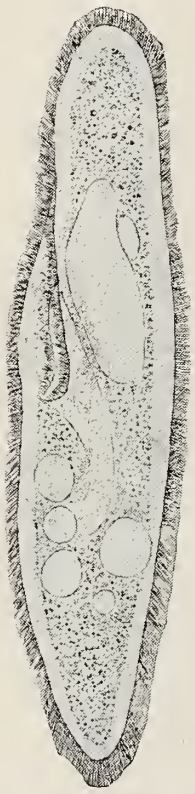


Fig. 4. PARAMOECIUM (Highly magnified)

group of tiny plants that includes many of the disease germs. Leeuwenhoek did not know that such bacteria cause disease. That was not found out until much later. But he did marvel that these tiny living things ate up the decaying bodies of the dead clams. He thanked a kind Providence that made these "wretched beasties" to act thus, else the canals of his beloved Holland would have been full of rotting clams. (Demonstration Appendix, p. 293.)

Now, when Leeuwenhoek was a lad, there were in England some men interested in scientific discoveries, men like Robert Boyle, Robert Hooke, and Isaac Newton, who used to meet to talk about the new discoveries in science. At first they called themselves the Secret College, for they feared if people knew what questions they were discussing, they might be arrested and possibly beheaded. But in 1666, when King Charles I was reigning, they changed the name of their society to the Royal Society. The king was interested in the things they talked about and gave them permission to meet openly.

A Dutch friend of Leeuwenhoek wrote to the English Royal Society and told them they should get this lens-maker to write them about his discoveries; so the secretary of the Royal Society wrote to Leeuwenhoek and asked him to tell them about his microscopes and the things he saw with them. Leeuwenhoek did this and for fifty years

he sent a fairly steady stream of letters and papers, 375 in all, to this Society about his discoveries. Many of them were published in the Proceedings of the Royal Society and others were kept in the Library of the Society. The descriptions and drawings are so accurate that we can to this day recognize many of the things he saw.

DO LIVING THINGS COME FROM THE NON-LIVING?

FOR a great many hundreds of years even the scientists believed that living things originated from non-living. Aristotle, the old Greek zoologist (Fig. 5), tells how to get a swarm of bees. When an ox dies, bury the dead animal, he says, leaving only the ends of its horns sticking out. Leave it alone for many months. Then cut off the tip of a horn and out of the hole will come the swarm of bees. If you go to a pond in early spring just when the ice is melting, you may see frogs crawling out of the mud on the bottom of the pond, so you seem to see the slimy ooze turning into frogs. After the heavy spring rains when the Nile valley is coated with mud, mice appear in countless

Fig. 5. ARISTOTLE

numbers. It was thought they just grew from the mud. If you let a piece of meat rot in the summer time, flies come crawling out of it, so the meat must have turned to flies. Such beliefs were common back in the times of Leeuwenhoek.

Some thoughtful people, however, were beginning to doubt the spontaneous generation of living things. In 1668, an Italian, Francesco Redi (Fig. 6), put pieces of fresh meat into several jars. Some of these jars he covered with fine cloth, like veiling, others he left uncovered. He saw flies go down into the uncovered jars, but none could get into the covered ones. He found that in time maggots (see Fig. 63, Life History of the Fly, for explanation of "maggot") appeared in the uncovered jars, and flies came from the meat; but this did not happen in the covered jars. So he concluded that unless the flies had a chance to lay their eggs on the meat, no flies would come from it. (Demonstration 2, Appendix, p. 295.)

Fig. 6. Francesco Redi. [Locy]

But when Leeuwenhoek discovered these very tiny living things, people thought surely such sim-

ple organisms might come into existence from dead matter. An Irish priest named Needham made some mutton broth. All the living things in it were killed when the mutton was stewed. Then he put this broth into bottles and corked them. After several days he found, under the microscope, that every drop of the broth was swarming with these tiny living things. He found you could do the same thing with pea soup or bean soup, so he thought he had proved that they did generate out of the non-living organic material.

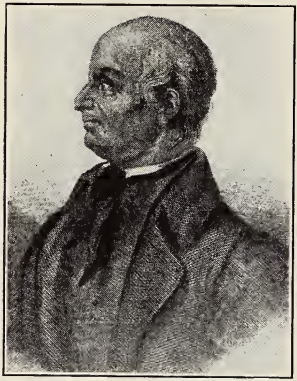


Fig. 7. Lazzaro Spallanzani [Locy]

But Lazzaro Spallanzani (Fig. 7), an Italian, a professor first at the University of Bologna and later at Pavia, was not so easily convinced. He put a drop of water full of these little "wretched beasties," as Leeuwenhoek called them, on a piece of glass. Beside this drop he put a tiny drop of pure water. Then with a fine brush he made a little water bridge connecting the drop of pure water and the other drop. He watched this under his lens until he saw one little creature swimming over to the drop of pure water. Then he wiped the bridge away and sucked up the water with

this one creature into a fine glass tube. He put it under his lens and sat down to watch it. He did not take his eye off the tiny animal. In about ten minutes he saw it divide into two, each just like the original. And in about twenty minutes more, each of these divided again and there were four little creatures. So he said they increase in numbers by dividing and not by spontaneous generation.

Then he repeated Needham's experiment, only he worked more carefully. He put his broth made from meat or from seeds into flasks with narrow necks. He heated the neck of the flask in a very hot flame until it melted. He drew out the neck into a small tube and closed this by melting it shut. He thought these tiny creatures might get into a bottle by squeezing past a cork that did not fit tightly, but he felt sure none of them could go through glass. He put these flasks into a bath of boiling water and left them there for an hour or more so as to kill all living things. After this he let them stand for several days. When he broke open the neck of the flask and examined the broth under his microscope, he found no living things.

But Needham said when you heat the broth for so long, you destroy the "vegetative force" in it and of course, living things cannot grow out of the non-living if this vegetative force is gone. So Spallanzani put seeds into pans and baked them

for hours until he felt sure the vegetative force was destroyed, if there were such a thing. Then he soaked these in water and let the stuff stand for several days in open flasks. He found the concoction swarming with tiny organisms. So he proved that there was no vegetative force to be destroyed by heat.

Then Needham said, "But when you heat your flasks you drive out the air in them so the pressure is reduced and the organisms will not grow without the normal pressure." So Spallanzani prepared his flasks again and sure enough when he broke the fine tube on the end of the neck, the air was drawn in. He could see it was, by holding a lighted candle near the opening when he made the break and the flame was drawn into the opening as the air rushed in. Evidently the pressure in the flask was reduced as Needham said it would be.

Next he prepared some flasks as before and drew out the necks into fine tubes, but he did not seal these at once. He left them open until the flask and the broth cooled off. Then he sealed the tiny opening by melting the tip of the fine tube in the flame, which took only a few seconds. He put the flasks with the broth in them into the boiling water, as before, to kill any living things in them. Then he let them stand. After several days, he broke the tips and held the candle flame over them as he did so. Now a little puff of air came out of the flask and blew the flame away from the opening. He

knew then that in these flasks, the air pressure had not been reduced. Yet he found no living things in the broth. You see how careful he was to try out every possible guess as to why these little living things did not appear in his flasks when they were properly sealed. He now felt that he had proved that living things, even these very tiny ones, did not arise from non-living stuff and that always such organisms came from other organisms of the same sort.

One would think that this question was now settled for good, but in 1774 Priestley discovered oxygen gas. He called it "dephlogistilated air." The name oxygen was given it by Lavoisier several years later. Priestley and others showed that oxygen is necessary for life. So some men thought that Spallanzani had driven the oxygen out of his flasks when he boiled the material or perhaps had changed it so it would not support life. A Frenchman, Pouchet, in 1859 made an experiment which he thought proved that these tiny living things did arise from the non-living. He filled a flask with boiling water and sealed the neck as Spallanzani had done in a very hot flame. When it had cooled, he turned it upside down and stuck the neck down into a large dish filled with mercury. He fastened the flask in this position. Then he broke off the tip of the neck under the mercury. The water, of course, still remained up in the flask. Next he produced some pure oxygen and led it from the

generator by a clean glass tube that opened under the mercury, just below the open mouth of the flask (Fig. 8). The oxygen bubbled up into the flask and drove out part of the water. So he had a flask half-filled with boiling water and half with pure oxygen, above the water. He then baked some hay in an oven until all living things in it were killed. He took little bunches of this hay and with forceps, heated to kill all germs on them, he introduced the hay into the mouth of the flask. When he let go of it, it floated up into the water. As it stood in the water it made a hay soup or hay infusion. In a few days, this was found to be swarming with tiny living things. It looked as if this careful experiment proved that living things did arise from the non-living. It was Louis Pasteur who took up the problem at this point. But we must see who he was and how he had been led to this problem.

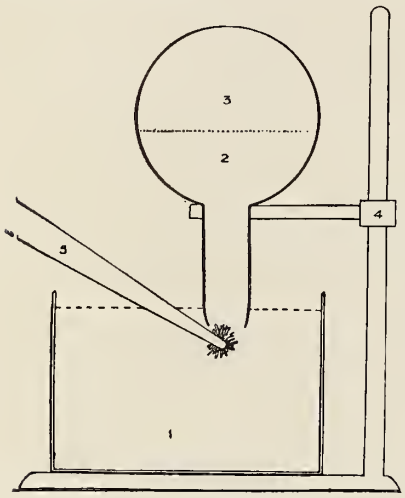


Fig. 8. DIAGRAM OF POUCHET'S EXPERIMENT.
A dish filled with mercury (1) has dipping into it the neck of a flask partly filled with boiled water (2) Above the water is oxygen (3). The flask is held in position by a support (4). The forceps (5) hold a wisp of sterilized hay to be released and float up into the water.

THE YOUTH OF LOUIS PASTEUR

Louis Pasteur (Fig. 9) was born at Dôle in the Jura Mountain region of east central France, December 27, 1822 (Fig. 10). His father, Jean Joseph Pasteur, when twenty years of age, was a soldier, in the armies of France. His regiment was called "the bravest of the brave" because it won several battles when the odds were fearfully against it. He was promoted to be a corporal, then a sergeant, and finally a sergeant-major and was decorated by Napoleon with the Cross of the Legion of Honor when he was twenty-three years of age. After the Napoleonic wars he took up the trade of a tanner, making leather from the skins of animals. When Louis Pasteur was a small boy, his father and family moved to the little town of Arbois, not far from where Louis was born. It was here that Louis spent his boyhood and attended school. He seemed merely an ordinary student, but he was known among his playmates as "Louis, the artist" because he was fond of drawing. Pastel sketches are still to be seen

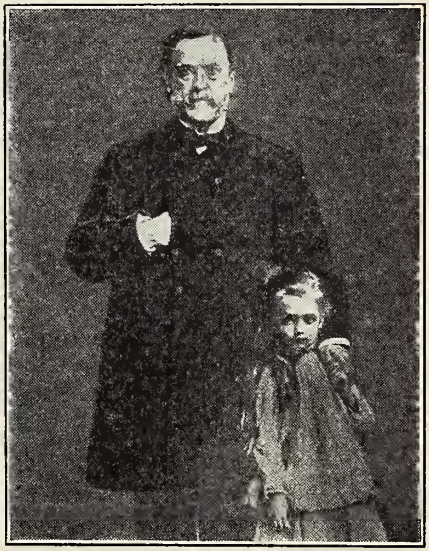


Fig. 9. Louis Pasteur and his Granddaughter [Locy]

which he made of his mother and father, the mayor, and other notables of the town.

It was the headmaster of the secondary school at Arbois who fired Louis with an ambition to go to Paris and attend the *École Normale* to prepare himself as a teacher of physics. So when Louis was sixteen years of age, he left home and traveled by stage to Paris (Fig. 11). But he was so homesick that his father came within a month and took him back home. He attended a college at Besançon, not far from Arbois, and after graduation, became a teacher in this school. He still had the desire to study in Paris, and so in 1842 he tried the examinations for admission to the *École Normale*. He passed them, but was fifteenth on a list of twenty-two who tried the examinations. He was not satisfied with these results and so the same fall he went to Paris to study and prepare for the examinations more carefully. He attended the *Lycée of Saint Louis* (Fig. 12). In 1843, he again tried the examination and

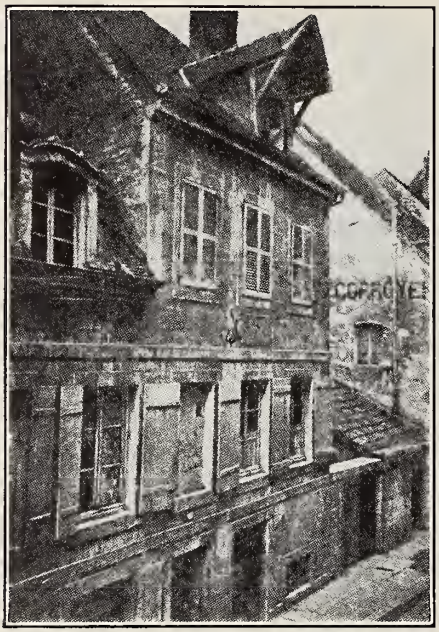


Fig. 10. Pasteur's birthplace [Keim and Lumet]

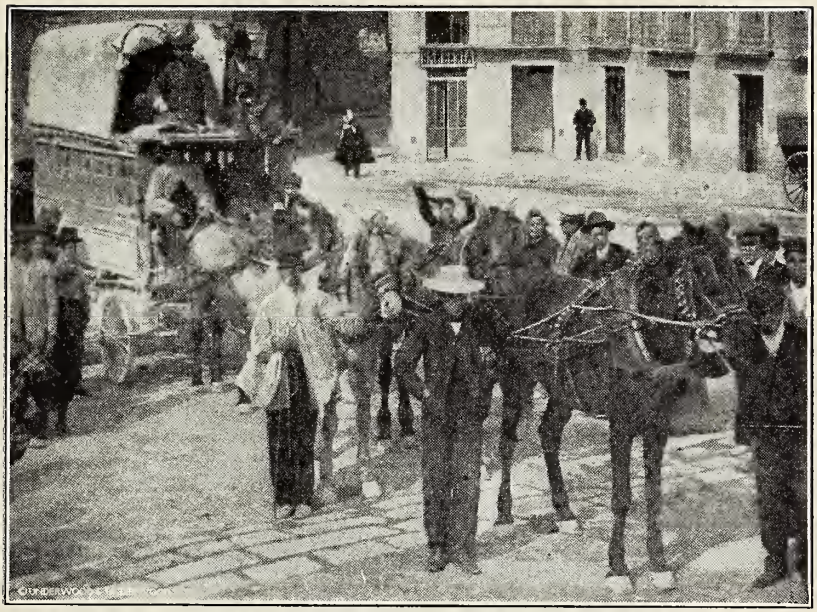


Fig. 11. An old-fashioned stage coach

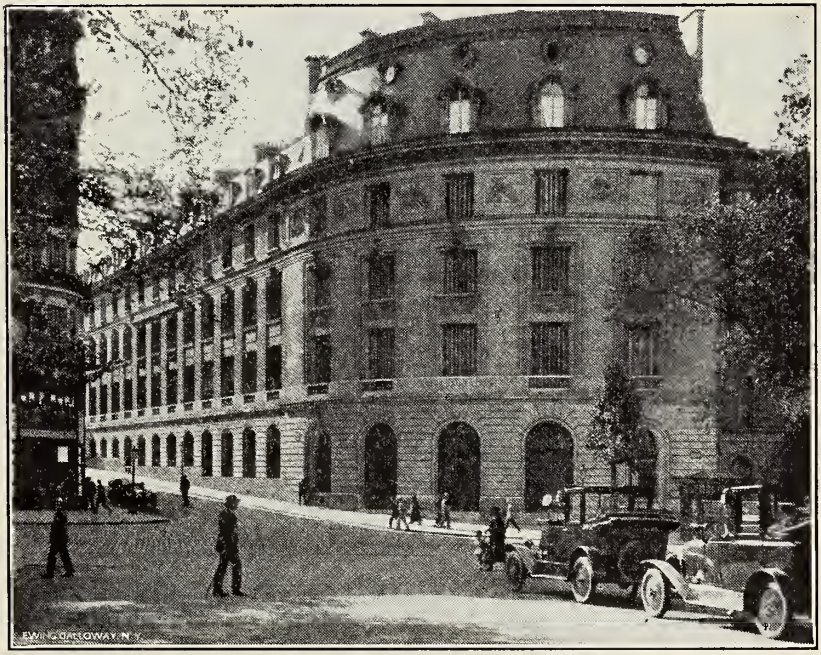


Fig. 12. Lycée Saint Louis, Paris

stood fourth on the list, and that fall he entered the *École Normale* (Fig. 13). While still a student, he continued teaching in one of the nearby preparatory schools, the one which he himself had attended, giving lessons in physics and mathematics. He also attended the lectures by Dumas on chemistry at the *Sorbonne* of the University of Paris (Fig. 14). He was much pleased with these lectures, which were very popular. Pasteur reports that it was necessary to arrive at the lecture room a half hour before the class began in order to get a good seat.

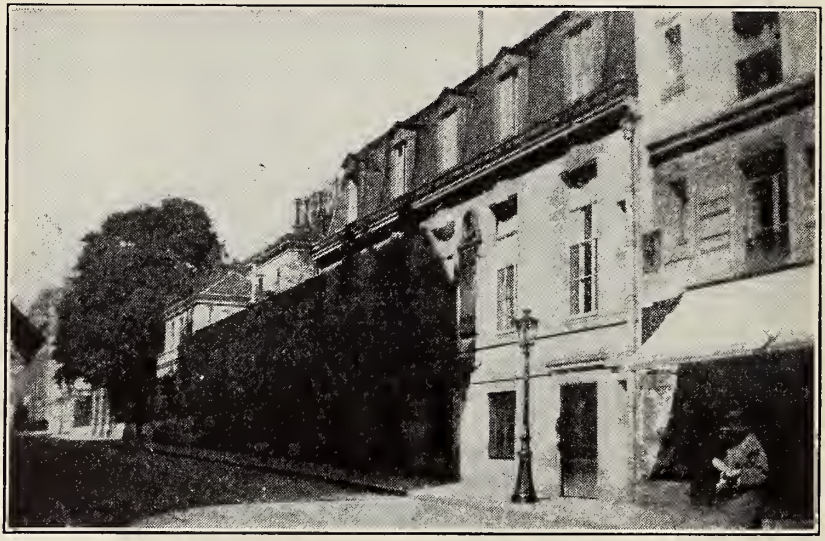


Fig. 13. Pasteur's old laboratory. *École Normale*

EARLY INVESTIGATIONS OF PASTEUR

In 1846 Pasteur was one of four out of fifteen candidates to pass the examination for teachers in the physical sciences in the secondary schools or

lycées. Then Boland, his professor, asked him to remain as his assistant in the *École Normale*. Pasteur accepted this offer gladly for it also enabled

Fig. 14. The Sorbonne and Rue Saint Jacques, Paris

him to continue his studies toward his doctorate. This higher degree he took in 1848. When a student is trying to obtain the degree of Doctor of Philosophy (Ph.D.) or Doctor of Science (S.D.)

he must make a real discovery. He must prepare a written statement telling how this was done and discussing how his discovery bears on other knowledge in this particular line. This statement is his thesis.

Pasteur's thesis was remarkable, for it announced a really great discovery. He had solved a chemical problem that had been puzzling the heads of older scientists for some time. Crystals of certain salts of tartaric acid form on the sides of wine vats (great tubs) when the wine stands in them for some time. Crystals are bits of solid substance, bounded by plane surfaces and are always of a definite and regular shape. A snowflake is made up of crystals of ice. When these crystals Pasteur was studying were dissolved in water and the solution was put into a polariscope (Fig. 15), the beam of light passing through the solution was sometimes turned to the right, and sometimes turned to the left. The cause of this peculiar behavior was unknown. Pasteur studied the crystals under his microscope and observed what had escaped the eye of previous students. While the crystals looked much alike, they were really of two sorts. These differed from each other much as one's right hand differs from his left. Each was the mirror image of the other. Hold your right hand up in front of a mirror and you will see that its image looks like your left hand. Carefully Pasteur sorted the crystals under the microscope and obtained a little heap of each

kind. When he dissolved the right-hand crystals in water and put the solution in the polariscope, it turned the light ray to the right. The left-hand sort, similarly treated, turned the ray to the left.

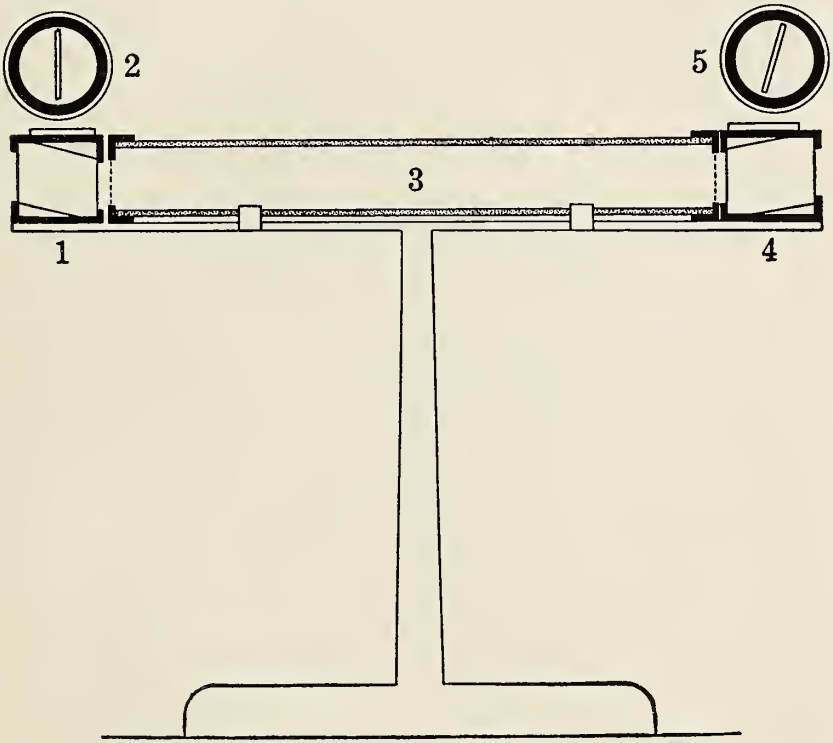


Fig. 15. Diagram of a polariscope. Light enters at the left through the crystal of tourmaline above 1. This crystal makes all the light waves move in one plane, as if all came out of the prism in the plane of the slit at left of 2. This light travels through the solution in the tube 3. If this solution is active the eye piece at 4 will have to be rotated as at 5 so as to let the light enter the eye looking in at 4, otherwise the instrument will be dark.

Now if equal quantities of the two crystals were dissolved together in water, the solution turned the ray neither to right nor left. Thus Pasteur showed that the study of crystal form could be

used to determine the chemical composition of substances. Boland, Biot, and Dumas, his teachers, were enthusiastic. They actually wept for joy. It was a real reward to have helped develop such a brilliant pupil.

Pasteur a Teacher

But now Pasteur must teach, and he went to the *Lycée* at Dijon as professor of physics. The following year he was appointed professor of chemistry at the University of Strasburg. Here Pasteur fell in love. Two weeks after he first met the young lady, he addressed to her father a letter requesting her hand in marriage. In this he said of himself, "I have absolutely no fortune. My only means are good health, some courage, and my position in the University." The young lady, Marie Laurient, was the daughter of the rector of the University. On May 29, 1849, the wedding took place. It proved a very happy marriage. Through a long and arduous life, Madame Pasteur was a rare companion and an able helper. Pasteur's great discoveries were made possible in no small measure by her great devotion and sympathy. No one gave this more generous recognition than did Pasteur himself.

In 1854, Pasteur was made a professor and dean of the new science faculty at the University of Lille. He had been continuing the study of the crystal forms of various chemical substances. He dis-

covered that when a solution of the two sorts of tartaric acid was allowed to ferment, only the right-hand sort was used up in the process. So fermentation enabled him to procure the pure left-hand form from mixtures of the two. It was while he was thus interested in fermentation that his attention was directed to a practical problem involving this process.

STUDY OF FERMENTATION

THE father of one of his students was manufacturing alcohol in his factory by fermenting beet sugar. But frequently the beet juice in the big vats would become sour and no alcohol would be produced. This man came to Pasteur to see if he could help him prevent this. So Pasteur went to the factory and secured samples of the material in the vats and took them back to his laboratory to examine under his microscope, the instrument that had helped him solve his previous problems. (Demonstration 3, Appendix, p. 295.)

No one knew at this time (1856) the cause of fermentation. The great chemists like Berzelius in Sweden, Liebig in Germany, and Dumas in France, thought it was simply a chemical action. The ferment, they said, was a decomposing substance that, by its breakdown, caused the fermentation. True, twenty years earlier it had been discovered that in malt, fermenting to beer, there

were tiny living things called yeast (Fig. 16), but the chemists doubted that these were alive. They thought they were merely forms of vegetable stuff that looked like living cells and were made in the process of fermentation.

But Pasteur found in the scum on the vats where the beet juices turned sour, many tiny things which he saw under his microscope increased in numbers as the souring went on. He found in the other vats where alcohol was being produced, much larger things that grew and budded and multiplied, though these too were too small to be seen except under his lens. If he transferred some of these things to a small quantity of fresh beet juice, the juice fermented and produced alcohol. If, on the other hand, he put some of the scum, made up largely of the tinier things, into fresh beet juice, it soured. He came to the conclusion after many additional experiments that the fermentation which produced alcohol was really due to the presence of the yeast, and that the tinier things, also living organisms, produced the acid that caused the souring.

Fig. 16. Yeast plant seen under the microscope

In 1857, when 35 years of age, he was recalled to the *École Normale* to be its executive head. His daily classes took up much of his time. His

administrative duties used up more time but he kept on with his investigations. His laboratory was an unheated attic, with only the crudest apparatus. He had no assistant. If he had not been a tireless worker he would never have accomplished all the tasks he set himself to do.

The next question to attack was—where do these living things that cause fermentation come from? The juice from the sugar beets was simply put into the vats and in due time it fermented, and alcohol was produced, or, occasionally, it soured instead. How did the yeast get into the beet juice? Whence came the other still smaller living things that caused the souring? Did they just spring up, living things out of the non-living? Were they thus spontaneously generated?

He knew that air is ordinarily full of tiny dust particles. If you let a small beam of light into a dark room, you can see these little specks of dust floating in the air. The beam of light seems full of them. Leeuwenhoek had put a drop of water full of the tiny living creatures on a clean glass. Then he set this away for several weeks. The water, of course, soon evaporated, but when he put clean water on the dirty spot again, the little creatures that had dried out, absorbed the water and began to swim around again. You know you can keep dried seeds for months and even years, but they will start to grow in a moderately warm place where they have air and moisture. Possibly

then, these tiny living things when they are dried out, float around in the air and resume living when they find suitable conditions. (Demonstration 4, Appendix p. 296.)

So Pasteur again made soup and put it into flasks. The necks of these flasks he drew out into long tubes, twisted several times like a letter "s" (Fig. 17). Then he boiled the soup in the flasks for a long time to kill all the living things in it. He let the flasks stand. The air he knew could get into the flasks, so there would be plenty of oxygen, but he thought that in going through these curved tubes it would pass so slowly that the dust particles would have time to settle on the walls of the tubes. Sure enough, the soup in these flasks remained free from the little living organisms. (Demonstration 5, Appendix p. 296.) By these experiments and by others, he convinced himself as Spallanzani had done that these tiny creatures only come from others of the same sort. But they can float in the dust of the air when they are dried out. It was this floating dust on the sur-

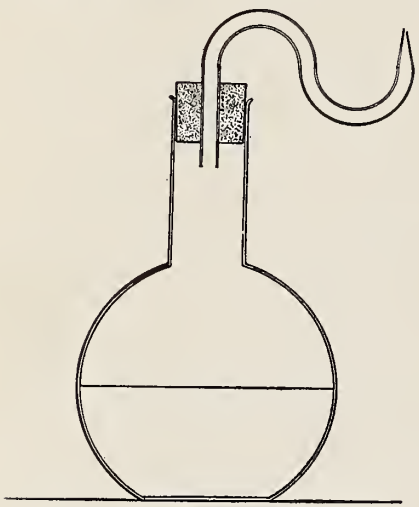


Fig. 17. Pasteur's experiment. He drew out the neck of the flask. In repeating the experiment the mouth of the flask may be closed by a rubber stopper, through which runs the S-shaped tube

face of the mercury that was caught in the hay and carried up into the water in Pouchet's experiment, p. 14.

PASTEUR STUDIES "SICK" WINE

ABOUT this time Pasteur was asked by some of the wine merchants to help them out of a difficulty. They told him that the grape juice left in great wooden barrels to ferment sometimes got "sick." Instead of fermenting to good wine, it would turn sour or become stringy or develop a bad taste. So Pasteur took samples of all these kinds of fermenting grape juice and examined them under the microscope. He found one sort of living thing, a yeast, growing in the juice that was fermenting properly, but in the other "sick" wines, he found different sorts of living things, one kind in the stringy wine, another in the bad-tasting wine. Moreover, he found that when he put some of the living things from the stringy wine into fresh grape juice, it would change into the stringy wine. The little organisms found in the bad-tasting stuff put into fresh grape juice, always produced wine with just this same bad taste. So Pasteur thought that wines become "sick" when the wrong sort of living thing gets into them. But that knowlege did not do the wine merchants any good. They wanted to know how to make the right ferment grow in the grape juice and how to keep the wrong sort from growing.

Now Pasteur knew you could kill all these tiny living things by boiling the liquid they grew in, and he wondered if some of them could be killed by less heat than was required to boil the wine. He tried it and found that if the grape juice is kept at a temperature of about 170° F. (the boiling point of water is 212°) for about twenty minutes the tiny living organisms are all killed. So he instructed the wine merchants to clean out the barrels thoroughly with boiling water; then when they were filled with the fresh grape juice to put in some of the juice that was fermenting properly and so plant the right kind of organisms. After it had fermented and produced wine they were to heat this to 170° and keep it at that temperature for about twenty minutes. They were then to bottle or barrel it in perfectly clean containers and seal these so none of the wrong sorts of ferments could get in. This the wine merchants did and found they had little further trouble with the wine becoming "sick." Now they could ship their wines to distant countries and it would arrive in good condition. Before this a cargo of good wine that started out from a French port might be found to be stringy or sour or bad-tasting when it arrived at the end of the voyage. Of course, it could not be sold. Now, however, by this new process they could treat the wine so that it would keep perfectly fresh. This process we now call pasteurization and it is used to kill the bacteria

so as to keep them from growing in many things besides wine.

SILK WORM DISEASES

And now came another call for help. Dumas, who had taught Pasteur his chemistry in his student days, begged him to go down to his old home in Southern France and find out how to prevent a disease of the silk worms. The production and manufacture of silk is one of the first industries of the region along the Mediterranean coast. A disease called "pebrine" was killing off the worms. The farmers who raised mulberry trees and fed the worms on their leaves were suffering because the worms died before they spun the silk. The factories where the silk cloth was woven had shut down for want of silk and the workmen and their families were starving.

Now Pasteur knew nothing about silk worms or their life habits, so he went to see Jean Henri Fabre, the great French naturalist, who lived in this silk-growing section. Fabre showed him the moths that laid the eggs from which the larvae or silk worms grow (Fig. 18). When first the worms hatch, they are very tiny, as big around as a hair and $\frac{1}{25}$ of an inch long, but they eat the fresh mulberry leaves from morning until night and grow very fast. In ten days or two weeks, they are as large as one's middle finger. Then each worm spins about itself a covering of silk.

This is produced by the worm itself from glands that secrete it. This silken covering, called a "cocoon," is about as large as the last joint of a man's thumb. Its walls are about as thick as blotting paper and about as firm. It is made of one strand of silk, wound round and round and back and forth. Inside of this the larva changes to a pupa. In about two weeks the pupa changes to a moth. It secretes a fluid that softens the silk so it can push the criss-cross strands apart and crawl out. Then the male and female moths mate and the female lays several hundred eggs that begin the whole process over again. (Demonstration 6, Appendix p. 297.)

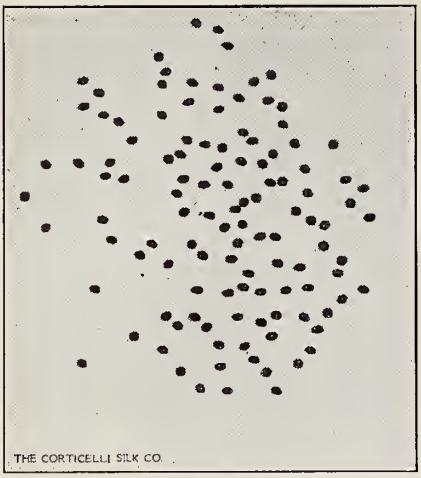


Fig. 18(*a*). Silkworm eggs

Fig. 18(*b*). Completed silkworm cocoon.

Fabre had all stages of this life history going on at his home so Pasteur could see it. The silk-growers sell most of the

cocoons to the factories, but of course, save some

Fig. 18(*c*). Silkworm feeding on mulberry leaf

to produce moths. But since one moth lays so many eggs, it is not necessary to save many. At

Fig. 18(*d*). Moths emerging from cocoons

the factory the cocoon is put into warm water to soften it, then the workman picks up one end of

the fine strand of silk and winds it onto a spool as it unrolls from the cocoon. These strands are then twisted together by a machine to make the silk thread and the threads are woven together to make the cloth.

Pasteur went down to Alais in the midst of the silk-growing country and found a room where he and his helpers could work. They secured both healthy and sick worms to study. Guérin Méneville had found, in 1849, that worms suffering from this disease, pebrine, had in their bodies many little transparent globules. Pasteur killed some of the sick worms, cut open their bodies and put some of the body fluid under the microscope. He, too, found these tiny transparent globules. Then he wondered if they were to be found in the moths, as well. So he examined them in the same way. In some he found many globules, in others none. Now he wondered if the moths that were infested with these globules were the only ones to produce larvae that became sick with the disease. When the moths had laid their eggs, he killed them and examined the body fluids. He even crushed their organs so he could examine the crushed material under his microscope. Thus he found that if a moth contained none of the globules, the larvae from her eggs would be perfectly healthy, but if the globules were present in the moth, the larvae from her eggs would sicken. They would grow some but soon they refused to eat the mulberry

leaves, their bodies became speckled with little black dots, as if they had been sprinkled with pepper. Then they soon died. He did not find this out all at once; in fact, he worked many months trying out first this, then that, to discover why the worms became sick.

He could work only a few months each year because it was only in the spring that the moths laid their eggs and the larvae spun the cocoons. Some of the cocoons were kept in cool places to be hatched out into moths the following spring. After working two seasons he thought he saw how he could help the silk-growers. He told them to wait until the moths had laid their eggs, then kill them and examine the organs inside their bodies. This was to be done under the microscope. If they could not find any of the little globules present, they could be sure that the larvae hatching from the eggs of such a moth would be healthy. But if the globules were present, then the eggs were to be burned for they would produce diseased larvae.

The growers at first thought they never could use a microscope for it was to them a strange instrument. But Pasteur taught some little children to recognize these globules in the crushed organs of the moth; then the growers realized that if children could do it, they surely could. And so as Spring came on and the moths began to hatch from the worms, they selected the eggs from moths that were free from the globules. But even then, many of

the larvae died. So many of the growers lost faith in Pasteur. They even talked of driving him out of the country.

But Pasteur, examining these larvae which had now died, found that they were infected with another disease. They did not develop the little black peppery specks on their bodies characteristic of pebrine, but all turned black. It was one of Pasteur's assistants, Gernez, who found that this disease was caused by another little living thing that grew in the digestive canal of the silkworm. It was more difficult to detect, but even that was possible. The worms were especially subject to it, if they became chilled or if the mulberry leaves on which they fed were not perfectly fresh.

PASTEUR IS STRICKEN

In the midst of all this hard work, Pasteur received word that his father was very ill. He hastened to his old home at Arbois, only to find that his father was dead. Shortly after this his family was traveling to Alais to join him when his older daughter was stricken with typhoid fever. Pasteur took his family back to Paris so she might have the best of medical care. He watched as a faithful nurse beside her bed for weeks, but finally she died. Then Pasteur himself was stricken with paralysis, a disease in which one loses the sense of touch or power of movement, or both, in some parts of the body. It was thought for a long time

that he would die but he felt he must live, he must solve this problem of the silkworms and save the silk industry in his beloved France. He must live to work out other problems he had in mind that he hoped would save much human misery. After months in bed he did recover, though not completely. His left hand was stiff and deformed. One leg was partially paralyzed so he walked with difficulty, dragging the left foot.

But in January as soon as he was able, he insisted on returning to his task at Alais. A few of the growers were willing to follow his methods. He himself, as soon as the moths began to hatch that Spring, examined thousands of them after the eggs were laid. Most of them he found diseased, but a few were free from the globules. The eggs of such were hatched, and the larvae tended, until they had spun their cocoons. Many of these he distributed to the growers so that they might have healthy moths for the coming spring. He was rejoiced now to receive letters from silk-growers in other countries, telling him that they had adopted his methods with success. There were enough growers now following his advice to begin to furnish an adequate supply of safe eggs.

SUCCESS AT LAST

THE next Spring Pasteur was offered the use of one of the old royal estates. Silkworms had been grown on this estate until the disease had killed

so many of them that the task became profitless. Pasteur took fifty ounces of eggs from moths that he had reared to start the silk culture again. The old buildings were thoroughly cleaned. The trays in which the worms were fed on mulberry leaves were whitewashed. All the tools were cleaned. Every vestige of the old stock of silkworms was destroyed. Pasteur gave half the eggs to the growers on the estate; half of them he took care of himself. Under his supervision, thousands of healthy worms were reared, spun their cocoons and twenty-thousand francs worth of the cocoons were sold that first year.

Meanwhile things were going very well in the south of France. The growers who had followed Pasteur's directions were successful. Other growers who saw their success became convinced, at last, that Pasteur was right. It was more than twenty years since this devastating disease had made its appearance in southern France. The one-time prosperous country was full of abandoned mulberry groves. The farmers were reduced to poverty. Many of the silk factories had been closed for years. But now once more prosperity was returning. The mayor of Alais voiced the thanks of the whole region and promised that in time, a statue of gold would be erected in honor of Pasteur's memory. This promise was partially kept. The statue that stands in the public square of Alais, an heroic figure of Pasteur, is not in gold,

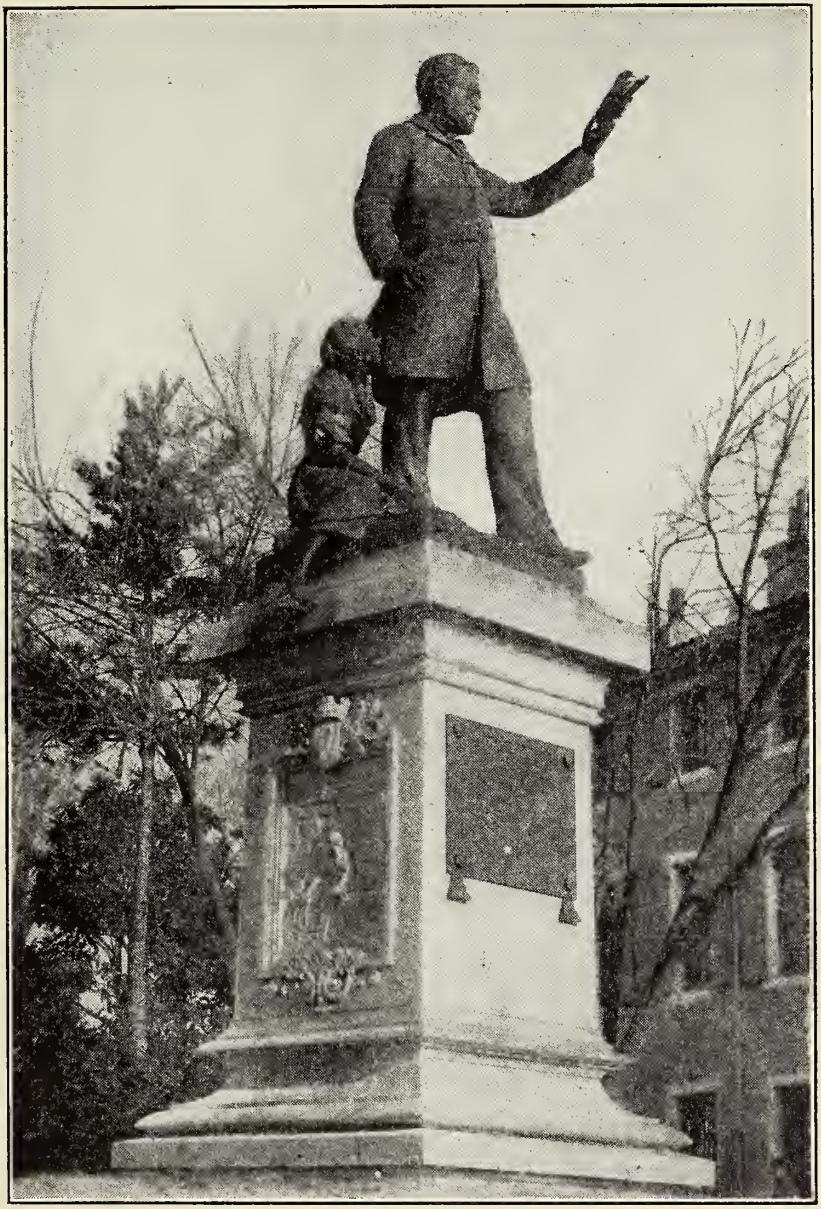


Fig. 19. Pasteur's statue at Alais

but it does commemorate the service of the man to this whole region of France (Fig. 19). It was estimated that the disease had cost the silk-growers 120,000,000 francs (about $24,000,000). It is no wonder that they were willing to express their thanks by a statue of Pasteur.

CHAPTER II

BACTERIA CAUSE DISEASE

OLD IDEAS

MANKIND HAD to wait for nearly two centuries after Leeuwenhoek first discovered bacteria in 1687 before they were shown to be the cause of disease. At first thought this seems strange, especially since Leeuwenhoek, himself, had shown that these tiny organisms were very abundant in the discharges of the body during certain diseases of the intestine. A few physicians of his time had even suggested that these bacteria were the cause of such diseases. But men were not ready to accept the notion. They still thought diseases were visitations of Providence, or that the sufferer was possessed of an evil spirit.

Even the doctors had no notion of the cause of the diseases they were trying to cure. True they could recognize many diseases and had learned the beneficial effects of some drugs. But frequently during the Middle Ages medicines were given for some fancied resemblance between the

remedy and the part afflicted. Thus the leaves of the liverwort, Hepatica (Fig. 20), were given for liver diseases, because the leaf of the plant is colored and spotted like the liver. The meats of walnuts were bruised and applied to the head to relieve headache because the shell of the nut is like the skull, its kernel is covered with thin coats like

Fig. 20. Hepatica or liverwort [Courtesy American Museum of Natural History, N. Y.]

the brain, and its meat is furrowed much like the brain. Vesalius (see p. 221), the father of modern anatomy, the science that describes the structure of the human body, died only sixty-eight years before Leeuwenhoek was born, and William Harvey (see p. 223), the father of modern physiology, the science that deals with the actions of the body

organs, lived at the same time as Leeuwenhoek. Thus medicine as a science was just beginning to revive after the ignorance of the Middle Ages.

PASTEUR'S DREAM

THE names of two men will always be gratefully remembered because of what they did to show that bacteria are the cause of disease. These men are Louis Pasteur, the Frenchman, and Robert Koch, a German.

Pasteur had now, through these many years of work, seen that tiny living creatures caused fermentation, that some of them are the cause of sickness in wines, others cause the diseases of silkworms. He had shown that these organisms never arise spontaneously, but always come from parents like themselves. He had proved that when dry some of them can float as dust in the air, but will grow and multiply when they find a suitable place that furnishes warmth, moisture, and food. He was beginning to wonder if germs might not be the cause of many diseases, and he was beginning to dream of the possibility of actually preventing disease. He had shown the silk-growers how to avoid pebrine and flachary, the disease of silkworms, the wine merchants how to prevent the sour, stringy and vile-tasting wines. Might it not even be possible to ward off human diseases? It was just a dream that he hardly

dared breathe to any one, yet the possibility took powerful hold of his imagination.

ROBERT KOCH AND ANTHRAX

But now we will leave Pasteur and his dream to note the work of another man, Robert Koch (Fig. 21). He was a young German doctor who had been graduated from medical school in 1866. He was a restless fellow, and moved from one village to another trying to find a good practice. It was in Wollstein, in eastern Germany, that his wife gave him a microscope on his twenty-eighth birthday. At first it was just a plaything and he amused himself evenings by looking at many objects under it. Then it happened that a bad epidemic of a disease broke out among the sheep and cattle of his neighborhood. This disease was known as splenic fever or anthrax. Animals struck by the disease refused to eat, hung their heads, stood with legs wide spread, as if afraid of falling, at last fell and died. When cut open,

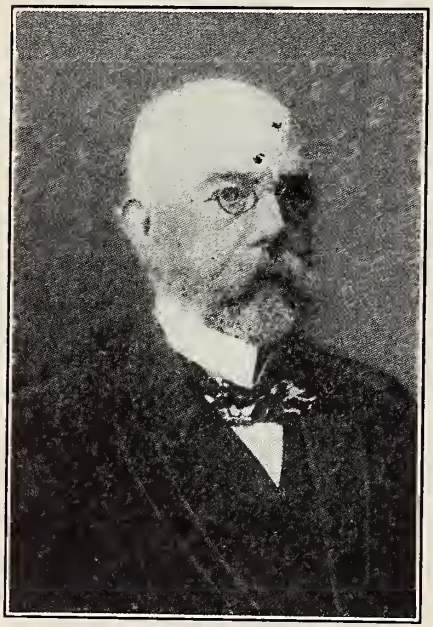


Fig. 21. Robert Koch. [Seelig]

their internal organs were found to be swollen and dark, especially the spleen.

Back in 1838, a veterinary, Detafond, had discovered that in the blood of animals dying of this disease there were to be found many little rods, so small they could be seen only under a microscope. In 1861, C. J. Davaine, a French doctor, suggested that these tiny rods which he also saw in the blood of animals with anthrax might be the cause of the disease. But he did not prove it. Now Koch went out to the farms where the disease was killing the sheep and drew blood from an animal that had just died. He put a drop under his microscope as soon as he returned home. There were the bacteria sure enough. He examined the blood from more dead sheep and in all that died of splenic fever he found these tiny rods. Then he looked at the blood of healthy sheep, dozens of them. He could find none.

But could he produce the splenic fever in well animals by putting into them the blood that contained the little rods? He was a poor young village doctor and could not afford to buy sheep with which to experiment, so he decided to try it on mice. They were plentiful enough. He baked in the oven some splinters of wood, so as to kill all germs on them. He held a mouse in his hand and made a little cut at the base of its tail. He dipped the tip of a splinter into the infected sheep's blood and stuck the point under the skin of the wound.

Then he put the mouse in a cage and waited. The next morning it was dead. He found its spleen black and swollen. When he crushed a bit of the spleen and put it under the microscope, there were thousands of the little rods and they were in its blood, too. Surely they must be living things that can grow and multiply, for he had put only a few of them into the mouse in the little bit of sheep's blood that stuck to the tip of the splinter. But here they were in every drop of the blood of the mouse and all through its spleen and other organs.

But after all, they might not be the cause of this disease. Perhaps there was something else in the blood that really made the mouse die. Could he grow these bacteria outside of the mouse and get them free of the blood? He thought they would have to be grown in some animal fluid. So he tried putting a tiny drop of blood containing them into a drop of the watery liquid from the eye of an ox. He put this under his microscope. But other bacteria from the dust in the air grew there, too, and he could not get a pure culture. Then he ground out a cavity in a piece of thick glass and polished its surface. He put a drop of fresh liquid from the ox eye on a piece of very thin glass, added a tiny bit of blood containing the bacteria and turned this piece of glass upside down so the drop was on its under side. He laid the thin glass on the thick piece, so the drop hung down in the cavity (Fig. 22). Next he smeared some grease around the

edge of the thin glass so no bacteria from the air could get in. He put this drop hanging in the glass cavity under his microscope. He could see in it many of the rod-shaped bacteria. He then put it into a box which he kept warm with a small lamp as he thought these tiny organisms would grow best if kept at about the same temperature as the blood in the living mouse. Once in a while during the evening he looked at it under the microscope and thought the bacteria were becoming more numerous, but he was not sure. He was up early

Fig. 22. A hanging drop culture
Section through the glass slide in which the hole is ground. This is covered by the thin glass on the under side of which hangs the drop

the next morning to examine it. Now there was no doubt. The drop was full of rods, not single ones, but long strings of them. He felt sure they were living things, growing and multiplying. (Demonstration 7, Appendix p. 298.)

Next he transferred some of this drop to a new drop of liquid from a fresh ox eye. He made the transfer on the tip of a needle. This he first heated red hot to kill all living things on it. In a similar way he kept this in the cavity of another glass strip and watched the few bacteria grow to countless thousands. He repeated this over and over, so he was sure there was left none of the blood used to plant the tiny rods in the first drop.

Would this pure culture give anthrax to a mouse? He tried it. He dipped the tip of a baked splinter into this last drop of fluid from the ox eye. He carefully stuck it under the skin in a little cut he made at the base of a mouse's tail. At the same time he dipped another splinter in fresh fluid from the ox eye in which no anthrax bacteria were growing. This he stuck in a similar way under the skin of another mouse. The mice were then put into separate cages. The next day the first mouse was evidently sick with anthrax and soon died. The other one lived, none the worse for the experiment. He tried this same thing again and again until he was sure it worked every time. Then he tried putting his culture into sheep in a similar way. They too died of the disease. He was now convinced he had proved that this little rod-shaped living thing was the cause of anthrax (Fig. 23). (Demonstration 8, Appendix p. 298.)

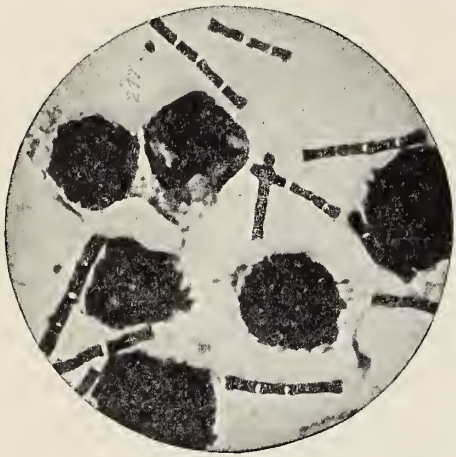


Fig. 23. The bacteria that cause anthrax [From Jordan's General Bacteriology.]

But still there was one puzzling thing. How did this bacterium get from one sheep to another? No one stuck blood-smeared splinters under their skins. Sheep or cattle would be kept for months

and months out of a pasture where some had died of anthrax. Snow and ice would lie on the ground all winter, rains would wash the soil. But when sheep were driven in to feed even a year or so later, the anthrax would attack them again.

Koch was sometimes called to see a patient while he was working on these bacteria. At several such times he found, on his return, that the drop of blood or the fluid from the ox eye that contained the bacteria had dried up. The bacteria seemed to have shriveled and died. It occurred to Koch finally to see if they were really dead.

He put a drop of fresh fluid on the dry spot on the glass, covered it carefully to keep out dust and put it in the warm box. The next day he was surprised to find that the drop contained many of the anthrax rods. Then he looked at a dried-up drop more carefully. True, the rods were shriveled and apparently dead, but in each were several tiny, shining granules. He covered the dry spot with fluid again and put it into the warm box as before. But every little while he took it out to watch it under his microscope. Then, to his surprise, he saw some of these granules change to the rods again. Perhaps the granules could live for a long time. He dried a piece of spleen from a mouse that had died of anthrax. He ground up a bit of this and examined it in water under the lens. No rods were present, but there were hundreds of the tiny, shining granules. He kept this dry

spleen for weeks and months. Then he put a bit of it into the watery fluid from an ox eye and set it in the warm box. In a day or two the drop was swarming with the rods. Probably these little granules could stand the snow and ice. So when the sheep came back to pasture after the winter was over, these granules would still be on the ground and would enter the sheep as they ate the grass.

Koch had been writing about his experiments to his old professor in the University of Breslau. Professor Cohn replied in encouraging letters. In 1876, Koch was invited to Breslau to tell them about his discoveries. Koch did more. He took his cultures and a cage of mice along. He repeated his experiments before these university professors. They were amazed, they were convinced. The news of the discovery went around the world. Koch was fast becoming famous. He had proved that one disease at least is caused by bacteria.

PASTEUR DISCOVERS HOW TO PREVENT ANTHRAX

PASTEUR had also been working with these bacteria of anthrax, for the flocks and herds of France were dying of the same disease. He, too, had grown cultures of them. He had produced the disease in well animals by injecting the cultures into them. But Pasteur was most anxious to help the farmers find a way to prevent the sheep and cattle from dying of the disease. He visited the

farms. One day he was walking in a field where were buried some sheep that had died of anthrax. He noticed that the ground over the sheep had a

Fig. 24(*a*). A bit of ground with many earthworm castings

different appearance from the rest of the field. Examination showed it was covered with the very numerous castings of earth worms. These worms eat the dirt and digest, as food, the particles of organic matter it contains. The rest is passed through their digestive canal and thrown out at the opening of their burrows (Fig. 24). (Demonstration 9, Appendix p. 299.) These burrows run deep into the soil, often three or four feet. Pos-

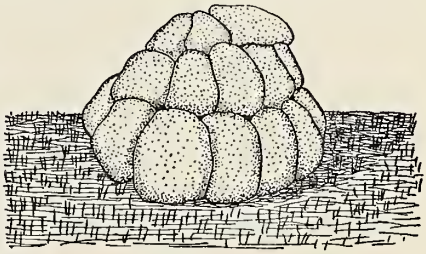


Fig. 24(*b*). Earthworm castings enlarged

sibly then these worms boring and feeding about the sheep would bring up the anthrax bacteria. He looked through his microscope at the castings mixed with water and found abundant bacteria. He injected such stuff into healthy animals and they sickened with the disease. He built a fence around such graves of dead sheep and put sheep inside the yards. Other sheep he put into similar yards in other parts of the pasture. The sheep in the pens over the graves died of anthrax. The others rarely did. So he advised the farmers not to bury animals, dead of anthrax, in the pastures, but to haul them to some dry sandy place or to a lime soil and bury them there. Earth worms do not like to live in such soils. This helped prevent the disease.

DR. KOCH PROVES TUBERCULOSIS IS CAUSED BY BACTERIA

Dr. Koch was now working on new experiments. He had been appointed by the government as director of a new laboratory built and equipped for his work in Berlin. He was provided with helpers and apparatus so that he could give his time to his research. The bacteria are so tiny and so clear that it is difficult to see them even under a microscope. So he tried to find dyes that would color them. He found that they would take certain colors and then stand out clearly under the microscope. More than that, some bacteria stained

deeply with one kind of dye, others not at all. These last might themselves stain intensely with yet another sort of dye. These stains then could be used not only to make the bacteria visible, but to tell one sort from another.

Koch now decided to try to find the bacillus of tuberculosis. No one knew there was such a thing. But Villemin, a French doctor, had just showed that tuberculosis was a communicable disease like anthrax. That is, one animal like a rabbit, or a guinea pig, that had the disease could give it to others kept in the same cage. It had been known for some time, too, that in the lungs or other tissues of animals and of people who died of this disease there were small, solid gray masses, like little beads. These were called "tubercles"; hence the name of the disease. Professor Cohnheim of the Medical School of Breslau had found he could plant one of these tiny fresh tubercles in the clear part of a rabbit's eye, when the animal was made unconscious by inhaling ether. Then when the rabbit recovered consciousness, Cohnheim could watch this tiny bit grow larger and larger and finally the animal would die of tuberculosis. Koch thought that these facts all tended to show that tuberculosis was probably caused by bacteria. But the task was to prove it.

He went to the hospitals when patients died of tuberculosis and examined the lungs and other organs of these persons after death. Always he

found the tubercles. He crushed these and smeared them on little glass plates, so that he could try to stain them with the dyes. After many failures he found one dye that would color a particular bacillus a bright blue but would stain other things only slightly. This bacillus would thus stand out clearly under the microscope from everything else so he could readily recognize it (Fig. 25). He found this particular bacillus always in the tissues of persons and of animals dying from the disease. He never found it in healthy persons or healthy animals. But this was not enough proof that it was the cause of the disease.

Fig. 25. The bacillus of tuberculosis [From Jordan's General Bacteriology]

He wanted to grow this blue-staining bacterium in pure cultures as he had the anthrax germ. Then if animals died of tuberculosis when injected with the culture, he would feel certain this one bacillus was guilty of causing the disease. He tried to grow it in the same fluids he had used for the anthrax germ, but it would not grow. He thought that since it grew in living tissues, it would have to be grown on something very like them. At last he

hit on a scheme that worked. He allowed fresh blood from an animal to clot. The clear fluid that was left he heated a little until it set as jelly does. On the surface of such blood-fluid gel he planted a few of these bacteria. To do this he heated the end of a wire red hot to kill all germs upon it. He dipped this end into a smear of the tubercle on a clean glass plate. Then he made a scratch on the surface of the blood-fluid gel, scraping some of the germs off from the wire into the gel. This gel was contained in a clean glass test-tube. The mouth of the tube was plugged with a wad of sterile cotton, which was removed only for a moment when the bacteria were introduced on the wire. (Demonstration 10, Appendix p. 299.)

Such test-tubes were kept in a warming box at about the temperature of the body. Day after day Koch watched to see if there were any signs of growth. Two weeks went by. Then he noticed little gray specks appearing along the line of the scratch (Fig. 26). These slowly grew larger. He lifted one of these out on the flamed wire tip and smeared it on a glass slide. Then he applied the dye and put the slide under the microscope. There were thousands of the little curved rods, each stained bright blue. Now he had found a culture medium on which he could grow these bacteria which he suspected were the cause of tuberculosis. He transplanted them from one culture tube to

another until he was sure that no trace of the sick lung tissue was left, so that he had only the suspected bacteria.

Next he stirred some of these bacteria from his culture into water and injected them into rabbits and guinea pigs. He waited to see what would happen. Weeks went by. But finally one by one

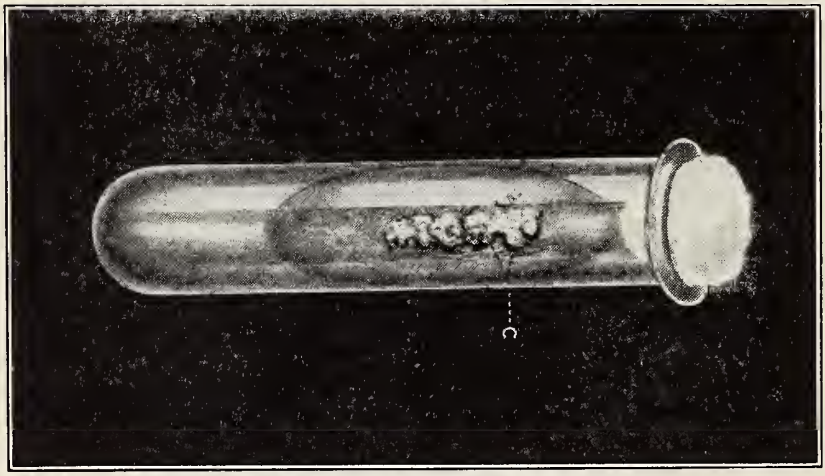


Fig. 26. Culture of the Bacillus tuberculosis
C, the colonies of the bacilli grown on the slanting surface of a jelly-like medium

the animals died. When he examined their lungs and other organs after death, he always found the little tubercles. These showed, when crushed and stained, the little bright blue rods—the bacilli of tuberculosis, now proved guilty of the disease (1881).

But was it not dangerous to handle these germs of tuberculosis? Suppose he had scratched his finger with the wire instead of the culture medium,

or had pricked himself with the sharp needle used to put the germs into the guinea pigs and rabbits. That was a risk he had to run. He felt certain the germs would grow in him and probably kill him if he should carelessly get them into his blood. He was careful to wipe off his hands frequently with chemicals that kill such germs; he kept his tools and glassware clean by boiling them in water. And luckily, he escaped. Not all men have been so fortunate who have studied these dangerous bacteria. Some have died in the midst of their studies, giving their lives, as we shall see, to help find out how to prevent other people dying from such dangerous germs. Dr. Koch is to be gratefully remembered as the first to prove certainly that a disease that attacks human beings is caused by one of these tiny germs, the bacillus of tuberculosis. Indeed, tuberculosis, until very recently, has been most dreaded, causing in civilized countries more deaths than any other disease.

JOSEPH LISTER DISCOVERS ANTISEPTIC SURGERY

Now let us turn to England and learn of some interesting things that were happening there. Joseph Lister (Fig. 27) was the son of an English merchant and was born in 1827. He was graduated as a doctor of medicine at the University of London. He went to Edinburgh as surgeon in a hospital and later (1860) became professor of surgery

in the University of Glasgow. Still later he moved to London.

The surgical ward of a hospital in those days was an awful place. When a limb had to be cut off or an abscess lanced, the wound often would not heal and the flesh around it would begin to rot. This was called gangrene. Shortly the patient died because the blood absorbed the poisons thus formed, or because the rotten blood vessels broke and the patient bled to death. The surgeon expected that more than half of the persons would die who had to undergo a major operation. And the worst of it was that no one knew why. Lister found the hospital wards none too clean. He had them scrubbed, kept the beds clean, and made the attendants wash their hands and clothing, often. That helped a little but not much.

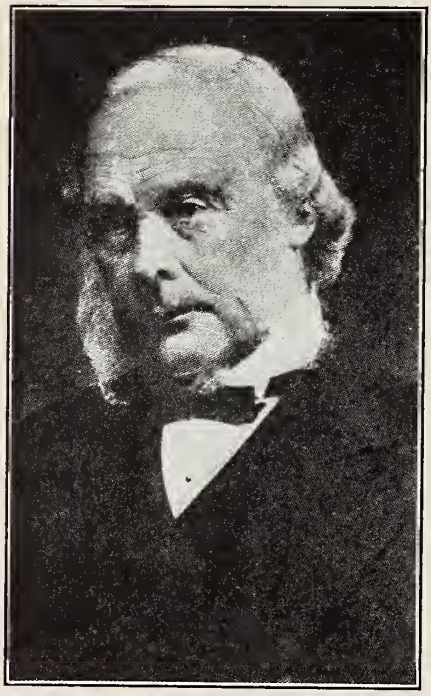


Fig. 27. JOSEPH LISTER [Masters]

Dr. Lister was wondering what more could be done to prevent such terrible suffering, and these many deaths in his hospital. What made the wound rot? He had been thinking about this a

long while when he happened to read (1865) Pasteur's paper on "Putrefaction." Remember how Pasteur showed that the air had floating in it many germs, dry bits of dust that dropped into the grape juice and, growing, made the wine sick. Pasteur had also showed in other studies that putrefaction or rotting was caused by tiny germs. If all germs in a substance were killed by heat and the germs floating in the air were kept out of it, the substance would not putrefy. That set Lister to thinking. Could it be that the rotting in a wound was due to germs that fell into it from the air? If this were true how could that be prevented? He knew that the city of Carlisle, near Glasgow, had used crude carbolic acid to destroy the bad smell of its sewage. The smell in the hospital wards was sickening, it was so vile. Possibly the bad smell of the sewage was due to rotting, too, and the carbolic acid destroyed the germs that caused it.

He decided to try out this new idea. One day a child was brought into the hospital with a broken leg, so badly fractured that the end of the bone stuck out through the flesh. Before, when such broken legs were set, in about seven out of every ten cases, the patient died. But now the wound in this child's leg was promptly covered with pads of cloth soaked in carbolic acid and was kept covered while the bones were set and while they knit. The patient recovered with no signs of gangrene.

So when Lister had to open an abscess and drain it, he sprayed the patient's skin over the abscess with dilute carbolic acid. He soaked his tools in the acid and washed his hands in it. Then he covered the abscess with cloths wet in carbolic acid and operated under this cover. He kept fresh cloths wet in the acid over the wound. You may imagine he watched that case very closely. Usually in such wounds, pus formed abundantly and then often the dreaded gangrene, or rotting, set in. But now very little pus formed. As day by day went by, the wound healed nicely and no gangrene appeared.

Time after time he operated and steadily the number of deaths after the operations became fewer and fewer as he became more skillful in keeping out germs. The fame of his method spread; many doctors came to watch him. They went back to their own hospitals to try his plan and found that it worked. Not only English doctors came, but many from foreign countries. Of course, some doctors thought it was all foolishness and that the old methods of operation were good enough. But their patients continued to die, so they were forced in time to adopt the new antiseptic surgery. It is a pleasure to note that the English people so appreciated this great discovery that Doctor Lister was made Lord Lister. (Demonstration 11, Appendix, p. 300.)

One never can tell how far-reaching a discovery

will be. Pasteur little thought when he was studying putrefaction and fermentation, finding out how to help those French wine merchants prevent their wines from going bad, that he was also helping to reduce the suffering in hospitals, and the number of deaths in operations. Lister wrote to Pasteur, "Permit me to thank you cordially for having shown me the truth of the theory of germs of putrefaction by your brilliant researches and for having given me the single principle which has made the antiseptic system a success." But Pasteur himself was helped by the discovery of an Englishman who had no notion he was going to help Pasteur. This man was Edward Jenner (Fig. 28) and the thing he discovered was how to prevent smallpox. Jenner died the year (1823) after Pasteur was born.

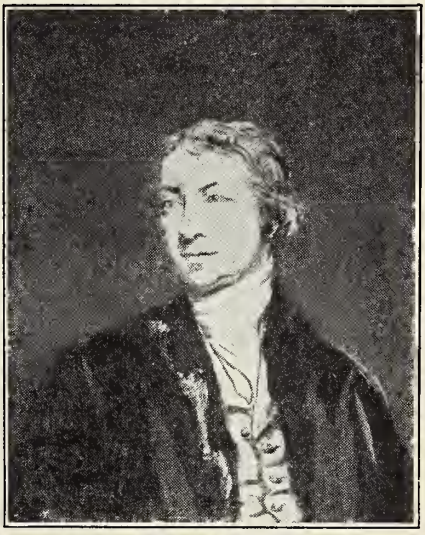


Fig. 28. Edward Jenner [Masters]

CHAPTER III

PREVENTION AND CURE OF DISEASE

EDWARD JENNER AND SMALLPOX PREVENTION

EDWARD JENNER was the son of a clergyman in the little town of Berkeley, not far from the large city of Gloucester, England. He was born in 1749. When a young man, he went to London to study medicine. He studied under John Hunter, a famous surgeon, who took a few young men as pupils. When he became a doctor, he went back to his home town to practice.

Now Jenner had heard, ever since he was a lad, that any one who had had cowpox would never catch smallpox. That was a common notion among the farming people. A cow with this trouble has little sores on her body, especially on the milkbag or udder. Often the milkmaid would catch this trouble and would have similar sores on her hands and arms. She might feel ill for a few days, but the illness was not serious. After this attack, the milkmaid never need fear smallpox, it was said. She might even take care of some one suffering with smallpox and not catch it.

Smallpox itself is a very serious disease. The persons who have it are very sick. They break out with small pimples full of pus, all over their bodies, but especially on the arms and hands. When these heal they leave scars or pockmarks. The disease was so prevalent in Jenner's day that six out of ten people you met would be pockmarked. About one-tenth of all deaths were due to this disease. Frequently it would become epidemic in a community, spreading from house to house. Nearly every one who had not previously had the disease would come down with it and people by the hundreds died of it.

Jenner as he went about as a country doctor began to ask people who had smallpox if they had ever had cowpox. He inquired of the milkmaids who had had cowpox if any had been sick of smallpox. He made hundreds of such inquiries and kept a record of the replies. He found that patients suffering from smallpox had never had cowpox and that those who had had cowpox never took the smallpox. For ten years he collected such facts until he felt sure that cowpox did prevent smallpox. He went to London in 1788 and told the doctors of his discovery. But they would not believe him. He could not prove what he said. So he went back home to collect more facts.

Finally, he felt so sure he was right that he decided to give his own little boy, then a year and a half old, the cowpox, so he would not run the risk

of catching smallpox. He made a little scratch on his arm and put into it some of the matter from a sore on a cow's milkbag. The little fellow had the cowpox, sure enough, but recovered. Then country people began to come to Jenner to be vaccinated with cowpox so as to escape smallpox. When smallpox broke out in their villages, these people always escaped. Jenner even tried to give smallpox to some of these vaccinated persons and could not, so now he felt he had proved that he was right. He published a paper about his discovery. Many doctors were convinced that Jenner's method was a preventative, and vaccination was practiced everywhere. Like all great reforms it met with much opposition. Some doctors even thought it was foolish. But the English people were so grateful that they gave Jenner, by vote of Parliament, $50,000 as a mark of their esteem and a token of their appreciation of his great discovery.

Abundant facts have proved vaccination a safe means of preventing smallpox.

PASTEUR DISCOVERS HOW TO CURE GERM DISEASES

Of course Pasteur knew about Jenner's discovery. Here was at least one case in which it was possible to prevent a human disease. Such prevention of disease was Pasteur's chief aim, the thing he was thinking about constantly. He felt quite sure that diseases were caused by these deadly germs, but how to prevent their action was the problem.

It was apparently a lucky chance that gave him the key to unlock the mystery. But as Pasteur once said, "Chance favors only those who are ready to profit by it."

Pasteur had been trying to discover the germ of another disease—the cholera of chickens, which caused great loss in the flocks of poultry on the farms. He found a tiny microbe abundant in the intestines of chickens that had died of the disease. It was not present in healthy chickens, so he suspected that this was the cause. He tried to grow it outside the chicken's body. But it refused to grow in the materials he had been using successfully in growing other microbes. Finally he made a broth of the gristle from chicken's flesh. In this the microbe grew successfully. He grew one lot of them after another, planting each fresh culture with a drop of the preceding one.

He used a little of this pure culture to inject into healthy chickens. They came down with the disease. He found the droppings from such diseased fowls were alive with this germ. It was easy to see then how the healthy chickens in the barnyard, picking their food off the soiled ground, would take the disease if one of the flock was sick.

Then one day in the midst of his studies he was called away and could not return for several days. Here on his table when he came back were some old cultures of the chicken cholera germ. He had left them uncovered in his hurry when he went away,

and they were partly dried out. But he thought he would try them anyway and see if they still worked. So he injected some into several chickens. They came down with the disease. He was called away again before he could wait to see what finally happened to the chickens. On his return he was surprised to find some of them alive and apparently quite well again. Evidently, he thought, the partly dry cultures are not so deadly as the fresh ones.

A few days later he wanted some more chickens to inject with fresh cultures. By chance his assistant brought in with the new fowls those that had recovered from the injections of the partly dry cultures. Pasteur gave them all the fresh germs and was amazed to find that the animals previously injected with the partly dry cultures did not get sick at all, while the others did. Here was the clue he had wanted for so long.

So he dried out some cultures for ten days, others for eight, and so on. Then he injected chickens with the ten-day old stuff. They were hardly sick at all. A few days later he gave them the eight-day old culture. There was no effect. A little later he used the six-day old culture with no effect, until finally he could inject the fresh cultures that would kill an ordinary fowl very promptly. But these chickens did not mind it at all. He had found out how to prevent the disease. Evidently some sort of resistance to the disease

had been built up in the chicken by the use of the stale cultures so they were not affected by the fresh deadly germs. It was something like preventing smallpox by giving the mild disease of cowpox.

THE DRAMATIC EXPERIMENT AT MELUN

NATURALLY the next disease that Pasteur turned his attention to was anthrax. In August, 1880, a young professor, named Toussaint, of the Veterinary School of Toulouse (a veterinary school prepares persons to treat the diseases of animals), claimed that he had found a way to prevent this disease. He took the blood of a sheep just after it had died of the splenic fever, separated the clot and heated the remaining serum which contained the bacteria and injected this into normal sheep. After this he injected the anthrax bacteria. He claimed that the sheep remained well. Pasteur showed that the bacteria of anthrax had not been killed by the heating but that they were weakened.

Meanwhile, Toussaint had tried another scheme of mixing the blood with carbolic acid. Again it was found that this merely weakened the bacteria. If either the heated blood serum or the blood mixed with carbolic acid were kept for a few days before it was injected into sheep, the sheep died even more promptly than usual.

Pasteur tried the same method of weakening

the anthrax bacteria that had proved successful in the case of the germs of chicken cholera. He found that if the cultures were kept in the laboratory and allowed to dry out partly for about twelve days, the deadliness of the microbes was so reduced that the sheep were only slightly ill. After their recovery they could be injected with more and more virulent cultures without harm. Pasteur felt so confident that he had discovered a preventative for this splenic fever that he undertook a demonstration on a large scale.

The Agricultural Society of the town of Melun put at his disposal a flock of sheep. Twenty-five of them were to be injected with the mild germs dried out for two weeks. A few days later more virulent germs were to be injected that had not dried so long and finally these twenty-five sheep, and another twenty-five that had not been treated, were to be injected with the perfectly fresh germs. The first part of the program was carried out on the fifth of May, 1881, in the presence of a crowd of doctors, farmers and veterinarians. The twenty-five sheep were each given five drops of the mild culture by injecting it with a small syringe (see Fig. 29) under the skin of the right thigh. Each sheep was then marked on its right ear so it could be told from those not inoculated. On the seventeenth of May each animal was given a second dose, but of the more virulent germs. On May 31st the final inoculation was made. When animals or per-

sons have such germs introduced into the body they are said to be inoculated.

Some of Pasteur's opponents were convinced that there was some trickery in this. One of them said that when Pasteur came to make the final injection he would use the top part of the liquid in the cultures in which there were no anthrax bacteria for the twenty-five sheep that had previously been injected, but that he would use the bottom part containing the deadly germs for the untreated

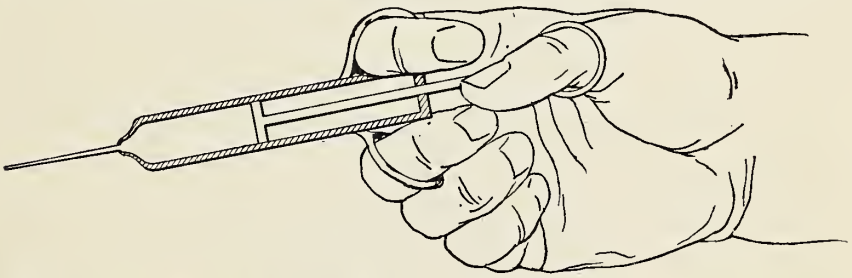


Fig. 29. Diagram of syringe, held by first and second fingers while the thumb operates the plunger.

sheep. But Pasteur let his opponents take the bottles containing the cultures and shake them up until they were satisfied that they were well mixed. Then his assistants injected the deadly germs into all fifty sheep.

Pasteur went back to Paris, leaving his helpers to watch the effects of the experiment He was so anxious, however, that he could hardly sleep, for he knew the test was a critical one. On June fifth he returned to the farm at Melun. Twenty-two of the unvaccinated sheep were dead, the others dying.

All had manifested the symptoms of severe splenic fever. The vaccinated sheep were all alive and well.

PASTEUR FINDS THE CURE FOR HYDROPHOBIA

PASTEUR was getting on in years, nearing sixty. He was suffering from the effects of that stroke of paralysis more and more, and his assistants had to do most of the experimental work for him, though his mind was clear and he could plan the experiments for them to conduct. But in spite of his handicaps he was now trying to decide what human disease he should attempt to prevent and he finally determined to study hydrophobia with this end in view.

His first task was to find, if possible, the little germ that caused this disease. He knew that the bite of a mad dog could give hydrophobia to another dog, or to a person. Naturally, therefore, he examined first the saliva from the mouth of the mad dog, but he found no germ present. He, himself, scraped the dog's teeth while his assistants held the animal and then he examined the scrapings but could find no germ that would cause hydrophobia. He examined the blood of the animal but still no germ. Since he knew that the mad dog finally died in spasms, that is, with violent contractions of the muscles, he next thought, that the nervous system was probably the seat of the disease. So he

examined under his microscope bits of the brain substance and the spinal cord, but still no germ was to be found. At last, he concluded that it must be so small that it could not be seen under the microscope.

Since he could not find any germ, he did not see how it was possible to make cultures, but he thought that if the virus was present in the nervous system, perhaps he could weaken it by the same drying process that had succeeded in the cultures of chicken cholera and anthrax bacteria. He found that if the spinal cord of a dog just dead of rabies were ground up and injected into the brain of a well dog, under an anaesthetic, of course, that this dog in time developed the disease; so he felt quite sure that he had located the seat of hydrophobia in the spinal cord.

He therefore took the spinal cord of a dog just dead of the trouble and cut it up into pieces. These he dried out, some for a day, some for two days, some for three days, and so on until finally the last of the pieces were permitted to dry for two weeks. He ground each of these up in glycerin, in which they kept well. The fourteen-day old stuff was then injected under the skin of a perfectly well dog. The next day it was given an injection of the thirteen-day-old material. So every day fresher material was used. Finally the dog was given an injection of perfectly fresh spinal cord from a dog

just dead of rabies. The result could not be known at once for it takes this disease about a month to develop. One may imagine with what interest Pasteur and his assistants awaited the outcome. Thirty days passed and the dog continued as playful as ever. As the days went by it manifested no signs of hydrophobia. Pasteur felt that he had achieved another success, but yet the experiments were tried over and over again so as to make them certain.

Still Pasteur hesitated to try out this means of prevention on the human being. But on the sixth of July, 1885, a nine-year old boy, Joseph Meister by name, was brought in. He had been savagely bitten by a mad dog. His mother begged Pasteur to try the remedy, for she knew the boy was doomed to a horrible death unless this new sort of vaccination could prevent it. So Pasteur agreed to try it. From day to day the boy received the injections and then came the period when all must wait to see if they were successful. Time came when the boy should have developed signs of hydrophobia, but none appeared. Apparently the method of prevention was as successful with human beings as with dogs.

Then came a second boy—a shepherd lad, Jupille by name. He had been severely bitten while keeping a mad wolf from attacking some younger companions. Again Pasteur and his assistants gave the remedy and again it was successful. This

lad, Jupille, later became an assistant at the Pasteur Institute.

THE PASTEUR INSTITUTE

THIS was Pasteur's crowning achievement. His fame was world-wide. From countries all over the earth, people, bitten by mad dogs, came to be cured. His little workroom was crowded to suffocation. Nineteen peasants came at one time from Russia. They had all been horribly bitten by a mad wolf. It was nearly three weeks after this before they could reach Paris, for travel was slow in those days. Pasteur and his assistants gave them two injections a day, morning and night, for the disease had a long start. All but three of the men recovered to go back to their homes as men who had risen from the dead. The world was deeply stirred. The Tsar of Russia sent Pasteur 100,000 francs to begin the building of a new laboratory. Money poured in from every country, and in due time, the Pasteur Institute was built (Fig. 30). In the yard about the building, in the midst of beautiful shrubbery, stands a statue of Jupille, defending a younger lad from the mad wolf. (Fig. 31.)

On Pasteur's seventieth birthday a great meeting was held in the *Sorbonne*, at the University of Paris. Noted men from all over the world were there to do him honor. On the arm of the President of the French Republic, Pasteur limped to the

platform. There were hundreds of students in the galleries. The whole audience rose and cheered while the band played the national anthem. Pasteur expressed his gratitude simply and then turned to the eager young faces—those students with whom as teacher he had spent so much of his life. "May you have," he said, "the immense

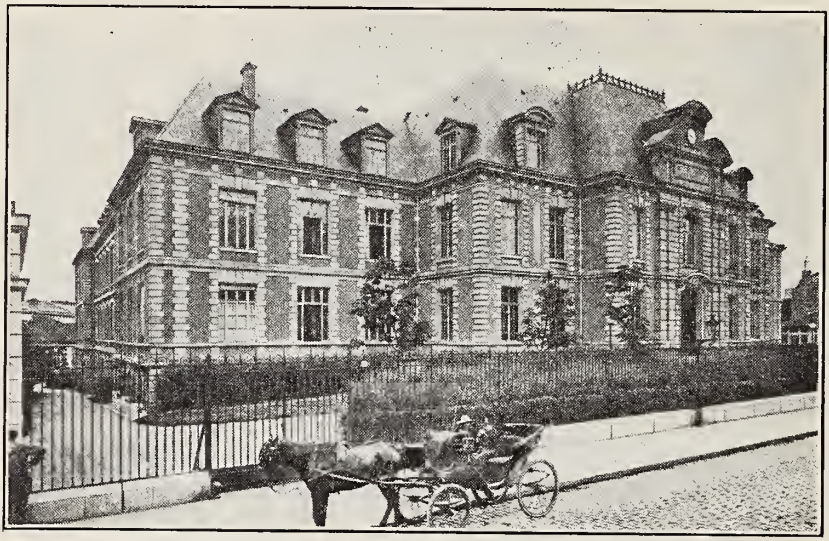


Fig. 30. Pasteur Institute

happiness of thinking that you have contributed in some way to the progress and good of humanity." That was his own greatest reward.

Sustained by this happiness, he died three years later, 1895, his hand in that of his faithful and devoted wife, his friends about his bedside. The French people, by popular vote, have proclaimed him their greatest and best-beloved man.

Fig. 31. Jupille's statue

THE WAR ON DIPHTHERIA

But the war against disease went grimly on. Pasteur and Koch had both been training able young helpers who were now ready to continue the work. From the Pasteur Institute and from the *Triangel* in Berlin, Koch's old laboratory, these men went out to discover other bacteria that cause disease. Koch himself and Gaffky, his assistant, Roux and Thuiller from the Pasteur Institute, went to Egypt to study Asiatic cholera, when an epidemic of that dread disease broke out. Later Koch went to India to continue its study. He found a bacillus, shaped like a comma, that was the deadly germ responsible for it. Loeffler and Behring in Germany, Roux and Yersin in Paris, were hunting for the cause of and a cure for diphtheria.

The method to be followed in such discoveries was now well known, thanks to Koch's work. First, must be discovered in persons or animals, sick of the disease, a bacterium that did not occur in healthy individuals. Koch's trick of finding some dye that would stain a particular germ but not other germs was a great help. Of course, the one stain for any given bacterium could be found only by many, many trials, when success came after hundreds of failures. Then this germ must be grown outside the person on some culture medium and kept in test-tubes in a warm box, an incubator. Koch, you will remember, had grown the anthrax

germs in the clear liquids from the eye of an ox, and the tubercle bacillus on a jelly made from blood serum. Pasteur had used a broth made from chicken gristle for the germs of chicken cholera. Finally, the bacteria so grown must produce the disease when injected into healthy animals.

Fig. 32. Edwin Klebs [Garrison]

It was Klebs (Fig. 32) who, in 1883, discovered the germ of diphtheria. The disease was raging then and many children were dying of it. He found a bacterium in the throats of such children which was shaped like a nine pin (Fig. 33). Loeffler, one of Koch's assistants, succeeded in staining it a bright blue with a certain dye (1884). He made a culture of it and injected the bacterium into rabbits and guinea pigs. They died of the disease, choking because they could

Fig. 33. The bacterium of diphtheria [From Jordan's General Bacteriology.]

scarcely breathe, their legs paralyzed; the same symptoms children showed who died of the disease. But Loeffler was perplexed. Sometimes he found these germs in the throats of healthy children. He never found the germs in any place but the throat. How could these bacteria, growing in the throat, cause paralysis? He thought that perhaps they gave off a poison—a toxin—into the blood and this was taken into the nervous system and so caused the trouble. He could not find the poison.

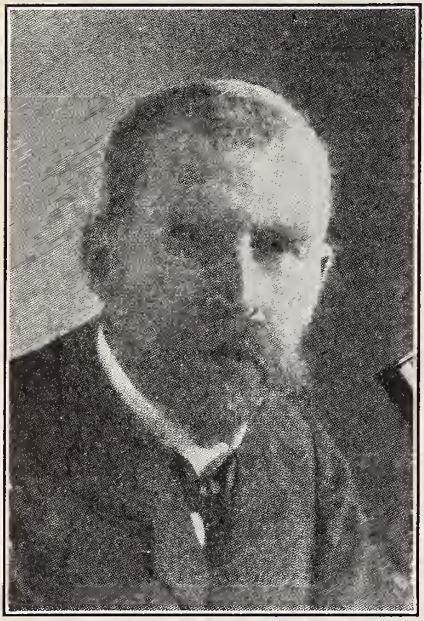


Fig. 34. WILLIAM ROUX [de Kruif]

But Yersin and Roux (Fig. 34) were more fortunate. After the germ Loeffler had discovered had been grown in the beef broth in the incubators for four or five days, they strained out all the germs by passing the broth through a fine porcelain filter. Such a filter is made of material like the dishes used on the table except that it is not glazed. The liquid, free from germs, should contain the poison, if there was one. This then they injected into rabbits and guinea pigs. But nothing happened. The animals remained perfectly healthy.

Perhaps, thought Roux, they were not injecting enough. So he used more and more. Finally when an ounce of the fluid was injected, the hind legs of the animals did become paralyzed and some of them died just as if they had been given the germs themselves. "But," said the experimenters, "surely this poison can not be very deadly if it takes so much. Perhaps we have not kept the germs growing in the broth long enough to produce much poison." So they left the cultures in the incubators for forty days. Then they filtered out the germs and injected the liquid. The guess was correct. Now it took only a few drops to kill an animal with all the signs of the deadly diphtheria (1888).

Recalling now how Pasteur had produced his weakened strains of anthrax and chicken cholera germs to use as a preventative of these diseases, scientists began to try something similar with the diphtheria germs. Hoffman tried old cultures; Frankel tried heat. Von Behring, working in Koch's laboratory, tried weakening the germs by treating them with various chemicals. In most cases the chemical killed the animal promptly when the culture treated with it was injected. But he kept trying, first one thing, then another. Finally, he hit upon a preparation of iodine. He mixed this with the germs and injected the animal with the mixture. In most cases, it failed too, but it worked in a few; several of the

guinea pigs that were very sick recovered after the injection of this chemical. He repeated the experiment again and again. Most of the guinea pigs died but always one or two recovered. That was hopeful.

Then he wondered if these recovered animals were still susceptible to the germs; so he injected a few of these recovered animals with fresh deadly germs of diphtheria—enough to kill an ordinary guinea pig very promptly. But these guinea pigs did not take the disease. The poison that the germs make must in some way be destroyed or prevented from acting at least. If the poison is in the blood, perhaps the antidote or substance that neutralizes the poison is there, too, he thought. He therefore drew some blood from one of his recovered animals. He let it clot so as to separate the clot from the fluid part. This fluid he mixed with a good dose of the diphtheria germs. He injected this mixture into a fresh, healthy guinea pig. He knew he had given it enough germs to kill it, yet it lived and showed no signs of illness. The fluid part of the blood (the blood serum) of an animal that has recovered from diphtheria must have something in it—an anti-toxin he called it—that destroys the poison which the germs of this disease produce (1890).

Now, would the plan work on other animals? He mixed the deadly germs with the iodine and injected the mixture into dogs and sheep. A few

days later he repeated the operation, using less iodine. He continued in later injections to reduce the amount of iodine used until the sheep were getting pure cultures of the diphtheria germs and still showing no signs of illness. From them he drew off fairly large quantities of blood. He removed the clot and kept the serum. Then, healthy sheep were injected with the diphtheria bacillus, enough to give them the disease violently. Immediately the new serum was injected. If this really did contain an antitoxin, it should neutralize the toxin produced by the diphtheria germs, so the sheep would not become ill. A second injection of the serum was given a day or so later. You may imagine with what interest he watched those sheep. Would they sicken and die, or would they live, and thus prove his idea correct? Day after day went by and the sheep remained quite well.

ANTITOXIN SAVES CHILDREN!

IN 1891 Von Behring was so convinced that the antitoxin in this serum of sheep's blood would cure diphtheria that he tried it on a sick child in the hospital in Berlin and it worked splendidly. Roux in Paris heard of his success and tried the method of developing antitoxin in sheep for producing it in horses. The horse seems to make the antitoxin in its blood very rapidly and in large quantities. It has so much blood that a goodly supply of the antitoxin serum can be obtained from

it—much more than from a sheep. He, too, tried this serum on children in a Paris hospital (1894). There was an epidemic of diphtheria in Paris and many cases in the hospitals. Roux gave them all the treatment, one injection of the serum followed by another within twenty-four hours. The results were marvelous. Many of the little sufferers with pinched faces and high fever were relieved almost at once. Some were too far gone to be saved. This particular children's hospital had a record of nearly four thousand cases of diphtheria and more than two thousand had died. The new treatment cut this death rate in half. Now the doctors do much better than that. If the antitoxin serum can be given promptly as soon as diphtheria is suspected, the patient is almost sure to recover. It can rarely save those in the late stages of the disease.

THE SCHICK TEST

In 1911 a German physician discovered a test to find out whether a person would take diphtheria if exposed to it. Some people seem to have a natural immunity to certain diseases. Pettenkofer, a German who did not believe in Koch's comma bacterium as the cause of cholera, swallowed a whole culture of these bacteria from a test-tube Koch sent him and was not the least bit sick. Jupille, however, at the Pasteur Institute, who took a very small dose of them, came down with

the disease. Schick's test consists in injecting a very tiny bit of the toxin-antitoxin under the skin. If this area becomes very red, the person is likely to take the diphtheria. If no redness appears, he is not likely to catch it; he is immune. Thousands of persons have thus been tested. Now, too, it is possible to inject the toxin and the antitoxin, the former a trifle in excess, and so make the blood of a person develop a good supply of its own antitoxin. This protects anyone for a time who is susceptible to the disease from catching it when exposed. Prevention is much better than cure.

Thus, there was worked out through the experiments of a number of investigators the modern method of preventing diphtheria. Death from diphtheria today is wholly unnecessary, for prevention is entirely possible. Such deaths do still occur in large numbers, but it is because people have not yet learned the facts given above, or else because, knowing the facts, they carelessly fail to take the necessary precautions.

Klebs first saw the bacterium of diphtheria. Loeffler found a specific stain to make it plainly visible. He also grew cultures of it outside of the body and gave the disease to healthy animals by injecting them with the cultures. He suggested, too, that death was due to a poison made in the body by these germs. Yersin and Roux proved that this poison or toxin was formed by the bacteria. Broth in which the bacteria had been

grown, when freed from the germs, killed animals when injected into them. The toxin in the broth produced all the symptoms of the disease. Von Behring discovered how to produce the antidote or antitoxin for this poison in the blood of animals and how to use the blood serum containing the antitoxin to cure the disease. Roux found the horse a favorable animal for the production of large quantities of the antitoxin serum. Schick discovered the test for susceptibility and finally Von Behring (1913) found out how to prevent the disease by injections of toxin-antitoxin. What a debt of gratitude mankind owes to all these patient workers for discovering how to prevent this dreaded disease!

Note, too, that these men did something more. They uncovered a new idea—that germs do not always kill directly, but may make a poison that causes death. Such diseases as gangrene, which Lister was fighting, or tuberculosis, whose cause Koch discovered in the *Bacillus tuberculosis*, are really parasitic; the germs actually feed on the body tissues. In tuberculosis the substance of the lung or other infected organ is actually eaten up by the bacillus. Now here was a new way in which these tiny enemies of ours could get in their deadly work. They could poison us.

No sooner do diphtheria germs in the throat begin to make the toxin, or poison, than our blood begins to make antitoxin to counteract it. The

human blood does this rather slowly, the horse's blood rather rapidly. So we have to borrow some antitoxin from the horse to use in our blood in order to neutralize the poison. By putting just a tiny bit of the toxin into the blood of a person, the production of antitoxin is stimulated. In time, enough is made to prevent any evil effects from diphtheria germs that may grow in the throat. We are then immune to the disease. Gradually this antitoxin disappears from the blood, so the toxin must be introduced again in the course of a few years, if one wants to keep immune to the disease.

THE WHITE BLOOD CELLS GOBBLE UP BACTERIA

We have also a natural means of defense against these tiny enemies of ours. There are in our blood many thousands of tiny living creatures called phagocytes (Fig. 35) or white blood corpuscles. (Demonstrations 12 and 13, Appendix, p. 302.) They are cells that can creep about like the very simple animals. They can escape from the tiny blood vessels through the small holes that are found in the walls of these capillaries (Fig. 36). Whenever bacteria get into our bodies and begin to cause trouble, producing irritation, the phagocytes

Fig. 35. A white corpuscle of the blood with large irregular nucleus. Ingested bacteria at right

rush to the spot and gobble up the bacteria and digest them as food. If the phagocytes can eat them up, the disease germs can do us no harm.

But these tiny bacteria grow and multiply very rapidly. One splits in half and there are two. Each of these repeats the act and there are four. This process is continued every few minutes under favorable conditions. It has been calculated that a single bacterium could give rise thus in a week's time to a mass of them as big as the world. Such a thing does not happen because

Fig. 36. Diagram of a portion cut from a capillary
A tiny tube made of curved plate-like cells with an occasional opening, *O*, between them through which white corpuscles can force their way.

favorable conditions do not last long and because there are hundreds of tiny creatures that eat bacteria as their main food. But disease germs can multiply in our bodies with amazing rapidity. We may take in several million of them at once in polluted drinking water, or on unclean food, or when some one coughs or sneezes in our faces. Then the phagocytes may not be able to eat them up as fast as they multiply. In such a case, we are likely to come down with a disease.

If you run a sliver into your finger, it is likely to have on it some bacteria. In the warm moist tissue under the skin these may multiply rapidly for there is plenty of food for them. The spot begins to be inflamed, a sign of irritation. The phagocytes creep out of the tiny blood vessels

Fig. 37. Elias Metchnikoff in his laboratory. [Keim and Lumet]

and seek these irritating invaders. The bacteria are multiplying. The phagocytes are gobbling them up. More phagocytes arrive. More bacteria are produced. The white blood corpuscles crowd in on each other trying to get to their food. They even suffocate each other. Many die from

overeating or are killed in the jam. You see under the skin this dead stuff as pus. You cut the skin, squeeze out the pus and put on a drop of iodine to kill the bacteria that are the cause of the trouble. You see here through the skin evidence of this battle going on between the phagocytes and the bacteria. In most infections the battle goes on deep in our bodies, where one can see no evidence of it. It was Metchnikoff, a Russian (Fig. 37), who discovered this action of the phagocytes. He spent much of his time at the Pasteur Institute working there at Pasteur's invitation.

The blood is a fluid that contains besides these phagocytes, or white corpuscles, tiny bits of living matter tinged red by a substance called "hæmoglobin." There are about 50,000,000 of these red corpuscles in every drop of blood in our bodies. Each red corpuscle of human blood is circular in outline if seen as you would see a watch when looking at its face. When you look at the edge, however, each appears hollowed in on face and back (Fig. 38). The red corpuscles carry the oxygen from the lungs to the working cells of the body, such as muscle cells and gland cells, and then carry the carbon dioxide given off by these cells to the lungs where it is discharged in the air we exhale. Both red and white corpuscles are short-lived for

Fig. 38. Red corpuscles of the blood as seen from edge at left. Face view at right

they work hard. They must be constantly manufactured in the body. The red corpuscles are produced largely in the bone marrow, the white corpuscles in the spleen and the lymph glands.

CHAPTER IV

INSECTS AS DISEASE CARRIERS

PATRICK MANSON

Let us read, now, another chapter of these strange discoveries that science has made in the interest of human health. This one deals with the part that insects play as disease carriers.

The first insect found to be guilty of carrying germs to infect man was the mosquito, and the germs he carried were those of elephantiasis. This is a disease common in the tropical countries, especially in Asia. The sufferer's legs and arms swell and the skin hangs in folds as it does on an elephant's hide. You see how the disease gets its name.

The man who discovered the facts in this case was Patrick Manson, who is called the father of tropical medicine. He was born in Meldrum, a small town near Aberdeen, Scotland, in 1845. As Patrick grew up, he decided to be an engineer, for he was fond of machinery. He went to work in the shops of his great-uncle. But he met

with an accident that strained his back badly so the doctors ordered him to bed and kept him there flat on his back for a long time and told him he must give up engineering—a great disappointment. To amuse himself he read books on zoölogy and botany. These interested him so much that when he recovered, he decided to go to the university and study medicine. What seemed to young Manson at the time a cruel fate, that kept him from his beloved machinery, gave to the world a great medical discoverer. He studied in Aberdeen and in Edinburgh to become a doctor.

In 1866 he went to Formosa, China, to take charge of a Chinese hospital at Takau. Here he had a wonderful opportunity to study many diseases new to him, malaria, leprosy, yellow fever, cholera, elephantiasis, and others. He came back home in 1874 on leave for a year to rest, for living in a hot climate is taxing for most northerners. While at home he read many of the medical journals he had not been able to get hold of in China. One thing he read especially interested him, that a Doctor Lewis had found small worms called "filiaria" in the blood of men. When he went back to China in 1875, this time to Amoy, he decided to look for them. He examined blood from the natives under his microscope and easily found these filiaria. In fact, he found that about one out of every ten of the population had this tiny worm in his blood—often as many as five hundred in every drop (Fig.

39). It was especially abundant in people suffering from elephantiasis. He thought probably it was the cause of the disease.

Then he began to wonder how it could get from the blood of a person who had the disease to one who did not have it. It must be spread from person to person, else it would not be so common. He had taught two of the native helpers how to take a drop of blood from a patient and examine it under the microscope for the filiaria. They became quite expert and were more successful in

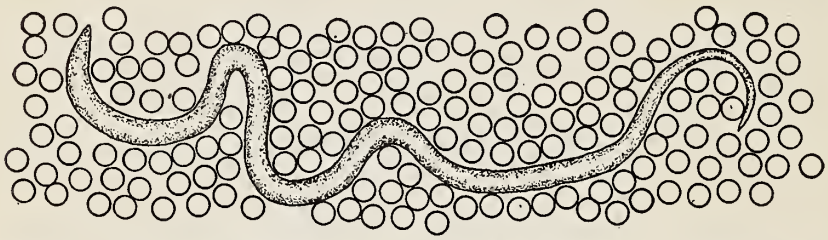


Fig. 39. Young filiaria worm among blood corpuscles. There may be hundreds of such larval worms in a single drop of blood

persuading the natives to let them take blood from them than was Dr. Manson. One of these assistants was on duty in the hospital by day, the other at night. When they reported to Dr. Manson, he was surprised to find that the man who worked by day rarely reported finding the filiaria, while the night man found many patients having them. Thinking this over he decided it meant that something that worked at night must be the agent that carried the filiaria from the infected person. This suggested the mosquito as the possible carrier

since it was very prevalent at night in the hospital wards, and not much in evidence by day. He then hired one of the hospital boys, in whose blood he found filiaria abundantly, to sleep in a room where there were mosquitoes. Early the next morning several mosquitoes gorged with blood were caught on the wall near the boy's bed. Dr. Manson examined their body fluids under his microscope and found them loaded with the filiaris worms. So he felt assured he had found the carrier. His proof was not complete for he did not actually give the disease to a well person by means of the mosquito's bite.

These little worms in the blood, it was found, are the young of the animal. The adult is three or four inches long and as large around as a horse hair. It lives in the lymph vessels that carry back to the veins the fluid bathing all the body tissues. These animals partly plug the lymph vessels so that the fluid, the lymph, unable to flow freely, accumulates in legs and arms and causes them to swell. The adult worm discharges its young at night, so the tiny worms are present in the blood in large numbers only then—a nice adaptation to the feeding habits of the mosquito.

THEOBALD SMITH DISCOVERS THAT TEXAS FEVER IS TRANSMITTED BY THE CATTLE TICK

A YOUNG American physician next showed that another insect was responsible for carrying a dis-

ease. Theobald Smith (Fig. 40) proved that the cattle tick (Fig. 41) was the agent that transferred the organism that causes Texas fever, a cattle disease, from a sick animal to a well one. And what in this case is most remarkable, the germ goes from the adult female tick into the eggs she lays and so into the young ticks. It is these young ticks that have never fed on a sick animal that cause the disease in the well animal.

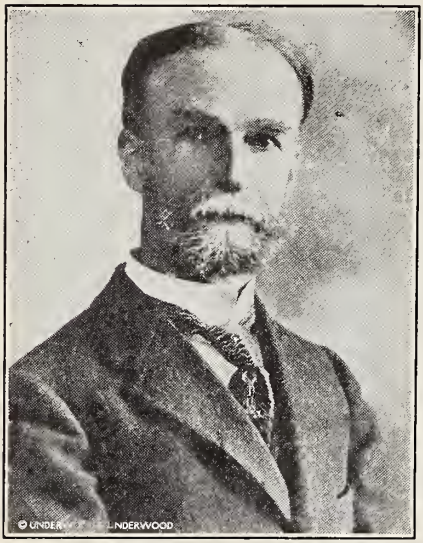


Fig. 40. Dr. THEOBALD SMITH, President of the National Tuberculosis Association

Theobald Smith was a doctor in the department of Animal Husbandry at Washington. He was an enthusiastic admirer of Robert Koch

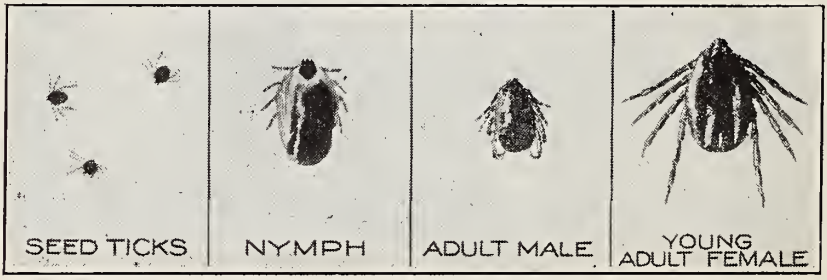


Fig. 41. The cattle tick young with male and female

and spent much of his spare time, nights, reading about Koch's discoveries. He did more than just

read; he tried to follow Koch's methods of making cultures. He studied bacteria under his microscope. He taught himself, at first in a crude manner, the way to rear and study the disease organisms. So when, in 1888, he started to unravel the mystery of Texas fever he was not wholly unprepared.

The cattlemen who raised the animals to sell for beef knew that if northern cattle were taken south, many of them died of this fever. They also knew that if cattle from the southern states were brought north and herded with northern cattle, these northern-bred animals caught the fever, sickened and died in great numbers, though the southern cattle were apparently perfectly healthy. The cattlemen, themselves, had an idea that the cattle tick was in some way responsible, but how they did not know. What puzzled them most was that if southern cattle, brought north, were turned out to pasture, the pasture became deadly. Ship the southern cattle to market, then put northern bred cattle in the pasture, even a month after the southern animals had gone, and the fever attacked and killed them. But this never occurred in the winter.

Now Theobald Smith had great respect for the hard-headed sense of the cattlemen. First he wanted to see if he could find any organism in the sick cattle that might be the cause of the disease, and second, if there were such, might it be trans-

ferred by the tick from one animal to another? He knew that ticks suck blood from the cattle and live on that food. So he naturally looked in the blood of sick animals for the germ. He was not long in finding a tiny thing in the blood corpuscles, so transparent that it looked merely like a hole (Fig. 42). These he never found in the blood of healthy animals. But he did find that the blood corpuscles of sick animals were destroyed in some way. Their blood was full of the wrecks of such corpuscles. Still that might be a sign of the disease, not the cause of it.

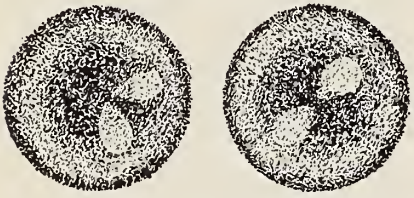


Fig. 42. The organism that causes Texas fever in the blood corpuscles from a cow

So he tried some experiments. He fenced off some small yards in a pasture. In one yard he put four cows recently arrived from South Carolina, where the cattle fever was known to prevail. He put some northern cattle in the yard with them. He hunted over three cows in the same shipment from South Carolina and killed all the ticks he found on them, big ones and little ones. He combed them thoroughly. He parted the hair of every part of their bodies to find the ticks. These animals hide among the hair, attaching themselves to the skin to feed and are difficult to find. But Smith and his helpers did a thorough job. These tick-free cattle were put in a second yard some

distance from the first and northern animals were pastured with them.

The cattlemen knew that northern cattle did not catch the fever at once. It was several weeks after they were herded with southern cattle that they would begin to show signs of the fever. So Smith waited patiently to see what would happen to the cattle in the yards. Two months went by. Then the northern cattle in the first yard began to sicken. They had a fever. They refused to eat. They stood around, with hanging heads, legs wide spread, and finally toppled over and died. But the northern cattle in the second yard kept perfectly well. The experiment was repeated again and again with the same results. It looked as though the ticks might be guilty. Then Smith drove these healthy northern cattle from the second yard into yard number one, where the tick-infested cattle had been. The southern cattle had been taken out and the dead northern animals removed; but the pasture still had many ticks in its grass. Here in time the northern cattle took sick and died.

Then Smith had grass, collected in the pastures in South Carolina, sent to him in big cans. There were plenty of live ticks in this when it arrived in Washington. He put these ticks onto northern bred cattle and watched them disappear in the hair of the hide. He and his helpers held the cattle so they could not brush the ticks off before they got a good hold on the skin. Again he waited.

In due time the cattle sickened of the fever and died. It seemed now that the ticks must be the agent that caused the disease, for in this experiment there were no southern cattle near from which the northerners could catch the fever.

HABITS OF THE CATTLE TICK

MEANWHILE Smith was studying the tick to learn its life habits. He found that the big females—a half inch long—after living on the blood of the cow for some time, dropped off to the ground and laid their eggs among the grass stems; then died. In time the eggs hatched into tiny young ticks (Fig. 43). Did the cattle then eat these eggs or the ticks on the grass and so catch the disease? He fed some of the grass sent north from South Carolina, ticks, eggs and all to northern cattle. But they showed no signs of the disease.

How, then, did the disease go from infected cattle to healthy ones? It was the end of the third summer of work and Smith was perplexed. He was not sure that the tiny creature he had found in the blood corpuscles of sick cattle was really the cause of the disease. He had apparently shown that ticks were the cause of the trouble, but how did they act to produce the disease?

Perhaps, he thought, the ticks just drain so much blood from an animal that it sickens and dies from loss of blood. This was a new idea and worth trying out. In his study of the life habits

of the tick, he had learned how to rear the young animals from the eggs. He had hundreds of such young in the attic room he called his laboratory. Cold weather was coming on. So he put stoves

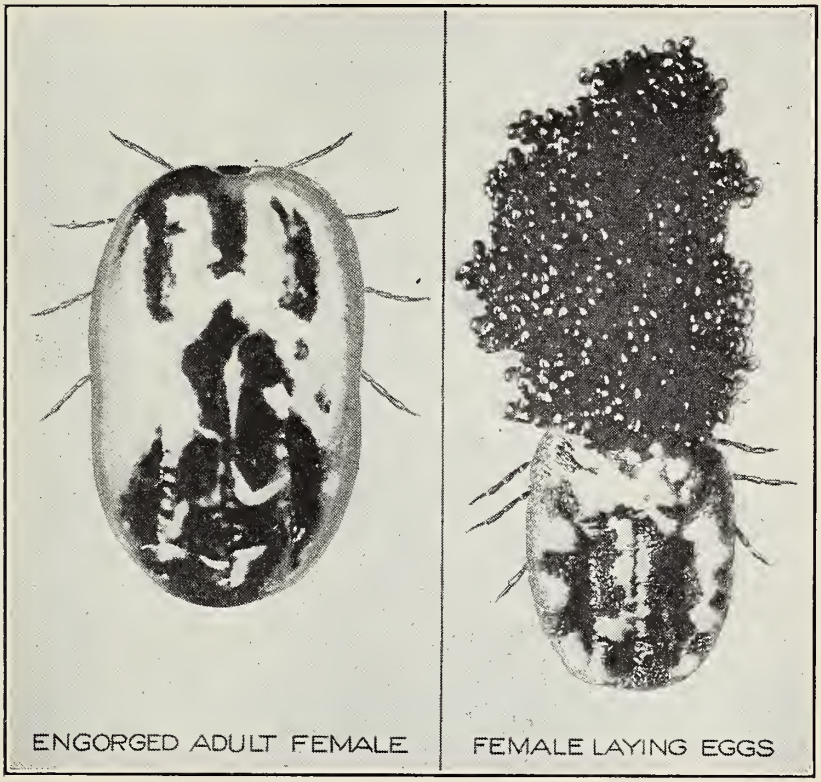


Fig. 43. Life History of the Cattle Tick. Female laying eggs from which "seed ticks" hatch, that later change to nymphs and then to adults. See Fig. 41

into a barn to keep it warm so the ticks would live. Northern bred cattle were kept in the stalls. He put the baby ticks he had reared on these cattle and watched to make sure they safely hid in the hair and secured a good hold on the skin before the

cattle had a chance to brush them off. He put on plenty of ticks so the animals would suck up much blood. He wanted the cattle to grow thin from loss of blood to see if they would show symptoms of the disease. Weeks went by. Then these cattle began to have a high temperature. The fever was coming on. They did grow thin, but it was evidently the disease that made them so. Smith examined their blood and there in the blood corpuscles were the strange little transparent organisms. Then the truth flashed on Theobald Smith. It was the young tick that gave the fever to the northern cattle, not the old ones. The blood parasites evidently went from the old tick into the eggs she laid and so into the young tick that hatched from the egg. Now it was plain why it took months for northern cattle to catch the fever from southern animals. The old females had to drop off from the cattle and lay their eggs. It took weeks for these eggs to hatch and for the young ticks to crawl up onto well animals and infect them by their bites.

But now, why do not the southern cattle, with plenty of ticks on them, have the disease? They do, as a matter of fact, for as calves they catch it in a mild form. Then those early mild attacks make them immune. Something in them is developed that prevents the organism from thriving in their blood. A few of their blood corpuscles are infected but not many; enough, however, to infect

the tick and its eggs and give the disease to northern-bred cattle. The latter have not developed this immunity when calves and so have the disease in a very virulent form. Theobald Smith and his helpers repeated these experiments to confirm the findings and it was not until 1893 that he published his results. The control of the fever evidently depends on the control of the tick. Now cattle and other live stock, like horses, that are subject to the disease, are given several times every season a bath in a chemical that kills the ticks on them without doing the stock any harm (Fig. 44). So even in the south where the disease was prevalent, it is being eliminated.

So the tick was proved to be a carrier of disease. Later, it was found that another species was guilty of carrying the organism that causes Rocky Mountain spotted fever—a disease that attacks human beings. It was another American bacteriologist, Howard T. Ricketts, who made this discovery and found a preventative vaccine for it. He began on this work in 1906. He went to Mexico City, in 1910, to study typhus fever. There he contracted this disease and died of it—another one of that long list of heroic scientists who have given their own lives to save the lives of their fellowmen.

MOSQUITOES AND MALARIA

MEANWHILE the mosquito was being proved to be the carrier of the tiny animals that cause malaria.

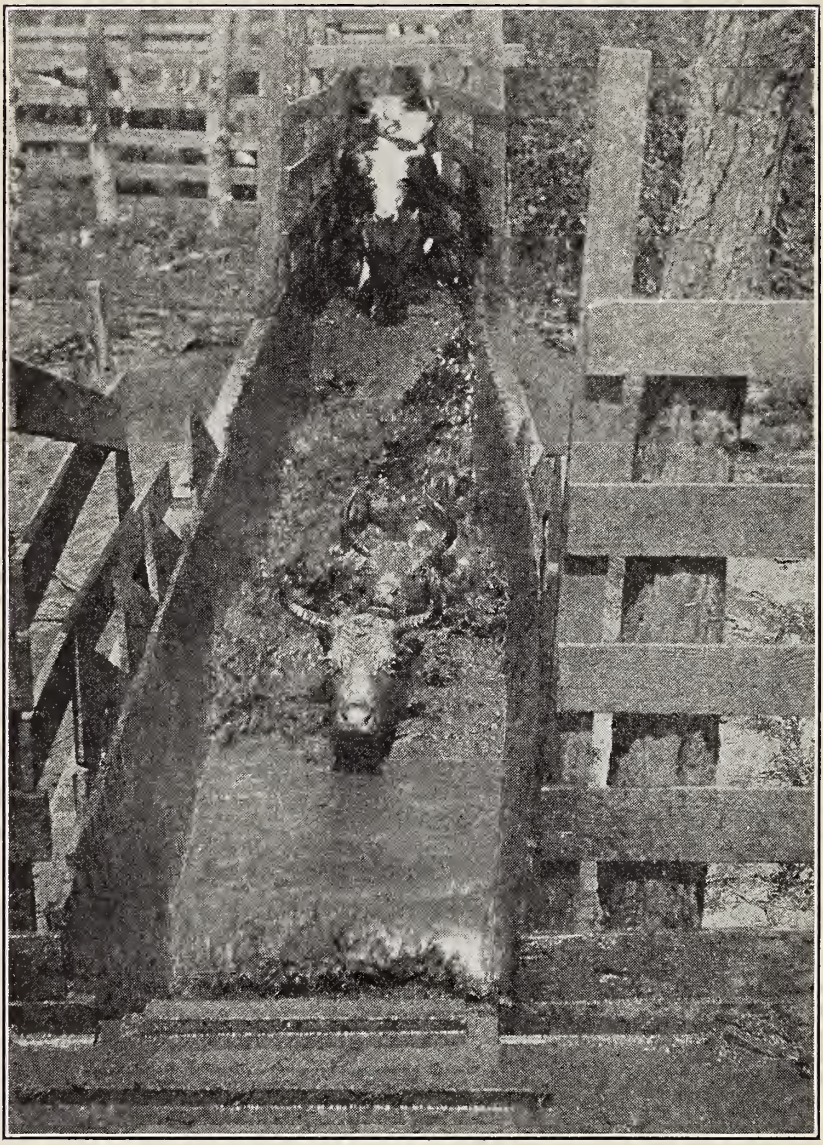


Fig. 44. Dipping cattle [Courtesy U. S. Dept. of Animal Husbandry]

As early as 1853 a French doctor, Beaupesthuy, suggested that malaria and yellow fever were both caused by the bite of the mosquito, but to the doctors of his day this seemed absurd. Several people, Merkel in 1847, Virchow, in 1849, and Frerichs, 1866, described and even drew pictures of a strange little transparent body found in the red blood corpuscles of persons that had malaria. Rasori, an Italian physician, suggested in 1846, that the spells of fever and ague that attack a malaria patient every few days are caused by the reproduction of some parasite in his body. But it remained for Laveran (Fig. 45), one of Pasteur's pupils, to put these things together. He was in Algeria in 1878; while there he found this little colorless object in the red blood corpuscles of persons suffering from malaria. More than that, he saw one of these little creatures throw out tiny whip-like lashes of its living substance and begin to swim about with their aid. He knew then that this tiny thing was really alive.

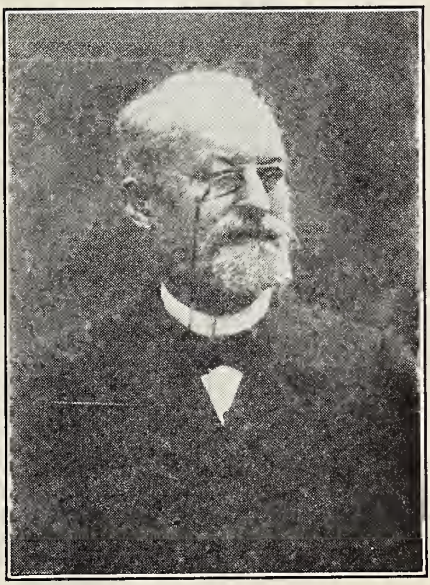


Fig. 45. Alphonse Laveran [Garrison]

This, he thought, was likely the parasite that causes the disease, as filiaris causes elephantiasis. He published his theory in the *British Medical Journal* in 1894.

Dr. Manson was then in England. He, himself, could not go abroad to study malaria, neither could Laveran. But Manson called Laveran's theory to the attention of Dr. Ronald Ross, of the Indian Medical service, who was then in England on a few months' leave. When he returned to India he began to hunt for the tiny parasite in all the organs of the mosquito's body. He spent two years at this job, dissecting hundreds of mosquitoes and examining every part. But he found no trace of the malaria germ. As a matter of fact, he had worked all this time on the wrong kind of mosquito. When he was about discouraged, he happened to get hold of two specimens of mosquitoes of the genus *Anopheles* (Fig. 46), the only sort in which the human malaria parasite lives. He even hunted through most of the organs of these without success. Then at last he discovered the little creatures in the walls of their stomachs. It was a long hunt. Most men would have been discouraged and would have given up. They would have concluded that the mosquito was not the guilty party after all. But to discover the mysterious ways of Nature requires just such dogged persistence as Ross had.

Ross hesitated at intentionally trying to give

malaria to human beings in order to secure the final proof that the mosquito was the real agent in spreading the disease. Dr. Manson wrote to him suggesting that he work on birds, as they also are subject to malaria. So Ross secured some birds suffering from malaria and allowed mosquitoes to bite them. He then put these mosquitoes into a

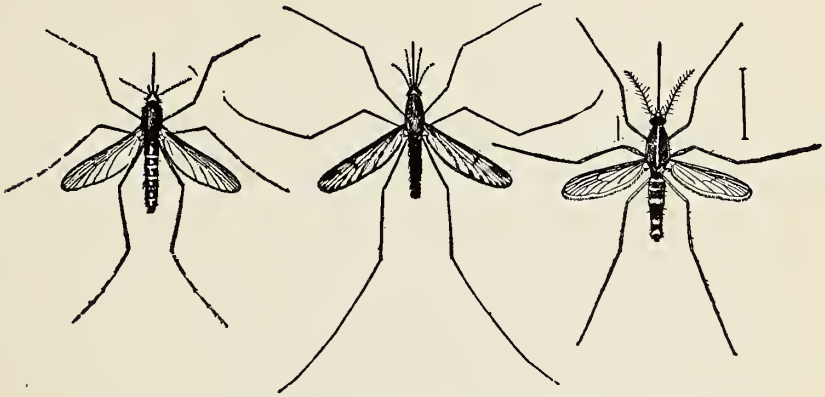


Fig. 46. Mosquitoes, all females

The one at the left belongs to the genus *Culex* and is a common northern mosquito that carries neither malaria nor yellow fever. The central insect is of the genus *Anopheles*, that carries malaria. The right-hand one is of the genus *Aëdes*, that harbors yellow fever. All greatly magnified. The short line at the right shows the real length of the insect beside it

cloth-covered cage with twenty-eight healthy birds and watched the mosquitoes bite them. All but five of the healthy birds came down with the disease. Ross reported the results of his studies to the British Medical Association at its July meeting, 1899. He himself could not attend but Dr. Manson made the announcement for him.

EXPERIMENTAL PROOF

Grassi, an Italian, who had read with great interest about Ross' work, induced four Italians living in Rome to let themselves be bitten by mosquitoes that had recently fed from a person suffering with malaria. All four came down with the disease. Then to make the matter doubly sure, Dr. Manson devised another experiment.

Up to this time it was generally believed that one caught malaria by breathing the night air in swampy regions. The swampy country around Rome was notorious for its malaria. It was always prevalent there. So Dr. Sambon and Dr. Low went from England to Rome. They built a little mosquito-proof hut out in the swamp country. Its windows and doors were double-screened; when they went out of the hut, they wore thick gloves and mosquito nets about their heads. They lived here for four months in the summer in perfect health in the midst of people suffering with malaria.

Then Dr. Bastianelli of Rome went to a pool in the swamp early in the morning and caught some mosquitoes that had just hatched out and so had not had a chance to bite any one. He took them in little cloth-covered cages with sliding bottoms to a patient of his who was suffering from malaria. He put the cages on the patient and slid out the bottoms. The mosquitoes lit on the sufferer's skin and bit him, gorging themselves with his blood.

The bottoms of the cages were then carefully slid into place again. These caged mosquitoes were sent by special messenger to Manson in London. His son and a Mr. Warren allowed themselves to be bitten by the mosquitoes. Both came down with the malaria, although the disease is unknown in England for the Anopheles mosquito does not live there.

LIFE HISTORY OF THE PARASITE

MEANWHILE Grassi was working out the life history of the parasite in the Anopheles mosquito and Ross was working out that of the parasite of bird malaria. This parasite is a tiny sphere of living substance that lives in the red blood corpuscle. When it has fed on this corpuscle and so killed it, the parasite breaks up into a number of still smaller bits, called "spores," each of which bores into a new red corpuscle. This process is going on in thousands of the red corpuscles at the same time. It is when the thousands of new spores bore into the healthy corpuscles that the patient has the chills and fever. When the parasite is taken into the stomach of a mosquito in the blood sucked from a person suffering from the disease, some of these spores produce male parasites, some female. After fertilization, the eggs (formed by the females), lodge in the cells of the stomach wall. Here in time they break up into hundreds of sharp-pointed slender rods. These work their way to the salivary glands.

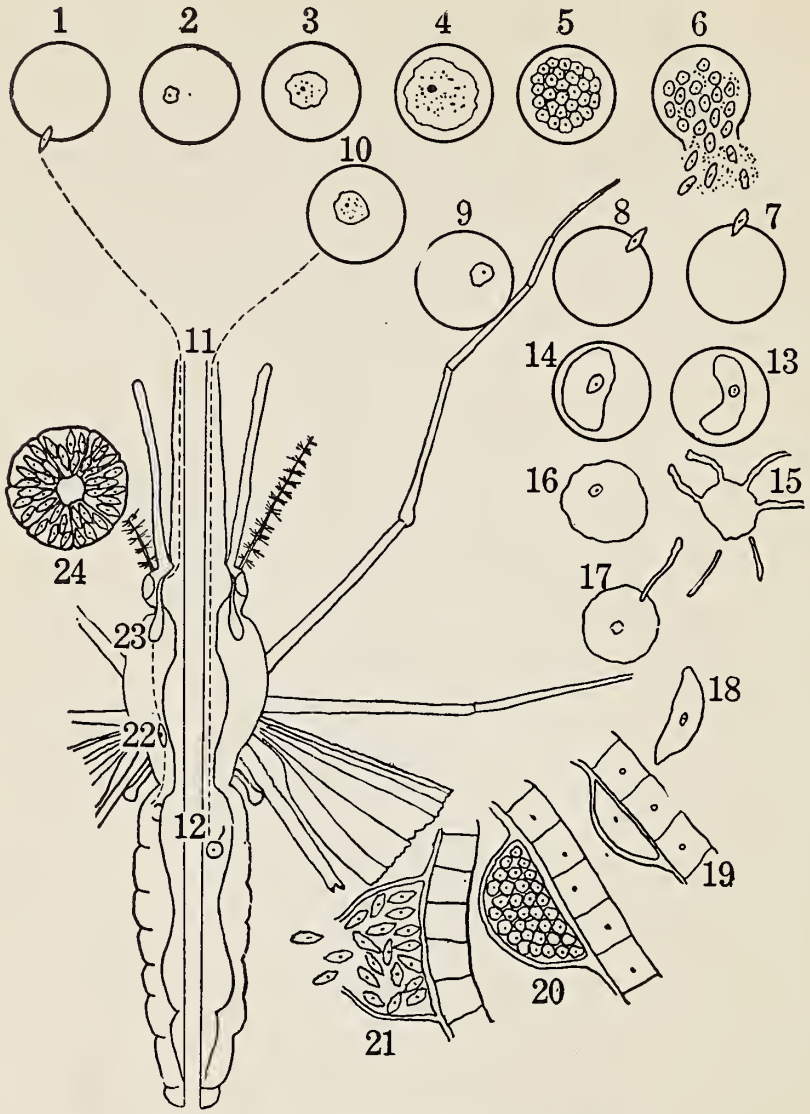


Fig. 47. Life history of the malaria parasite

1. The parasite forces its way into a red blood corpuscle where it grows, feeding on the blood corpuscle, 2, 3, 4. It breaks up into many small bodies (spores), 5, which escape, 6, as the corpuscle breaks. Each of these seek and enter a new blood corpuscle 7, 8, grow, 9, 10 and the process described is repeated, 3, 4, 5, 6 until many thousands of corpuscles are infected. When these break simultaneously, 6, and the millions of spores bore into new corpuscles the infected person has the chills and fever characteristic of malaria.

When the mosquito bites a man, the saliva is injected into the puncture to keep the blood a liquid while it is being sucked up into the mosquito's stomach. The rods or filaments thus get access to the victim's blood, bore their way into red corpuscles and so start the infection in the person (Fig. 47).

Dr. Grassi, the Italian, showed that it is only the female mosquito that feeds on human blood. The male lives on plant juices as does the female also much of the time. Blood seems to be necessary to her for the production of fertile eggs. The female mosquito standing on some floating object or close to the water's edge discharges a number of eggs into the water. These eggs are tiny spindle-shaped objects. They stick together and float as a little raft on the water. They are laid in ponds and ditches, in rain barrels, in swamps, in

———*Fig.* 47 *continued:*

If now an Anopheles Mosquito sucks up the blood from a malarial person, the corpuscles with the parasites in them (like 10) are taken up by the insects' sucking tube, 11, and carried down (right of 11) to the stomach, 12. (Right and left halves of the insect are separated somewhat to avoid confusion of ingoing and outgoing parasites.) In the stomach the parasites in the corpuscles grow, 13, 14, eat up the corpuscles and assume two different shapes, 15, 16. A detached portion (male gamete) of 15 unites with 16, the female gamete as shown at 17, also right of 12. After the union, 18, the new individual pushes its way through the wall of the mosquito's stomach, 19, and lies in a swelling on its outside. Here it grows, breaks up into many small individuals, 20, which discharge, 21, as the swelling ruptures. (See also left of 12.) These now travel, 22, to the salivary glands, 23, which they enter by the hundreds. A cross-section of a portion of the salivary gland is shown at 24.

When such a mosquito "bites" a person the sucking tube is thrust down through the skin. Saliva is then injected to keep the blood from clotting as it is pumped up as food for the mosquito. So the parasites get into the blood (left of 11) and find their way to the red corpuscles, 1, and the story starts over again.

fact everywhere where a bit of stagnant water stands. An old tin can may harbor them or a cupful of water in the hoofprint of a horse in the meadow (Fig. 48).

In the course of a week the eggs hatch into tiny worm-like larvae. These are the familiar "wrigglers" or "wiggletails" that swim in stagnant water, curling their bodies up like an S, then twisting the other way, a reversed S. Much of the

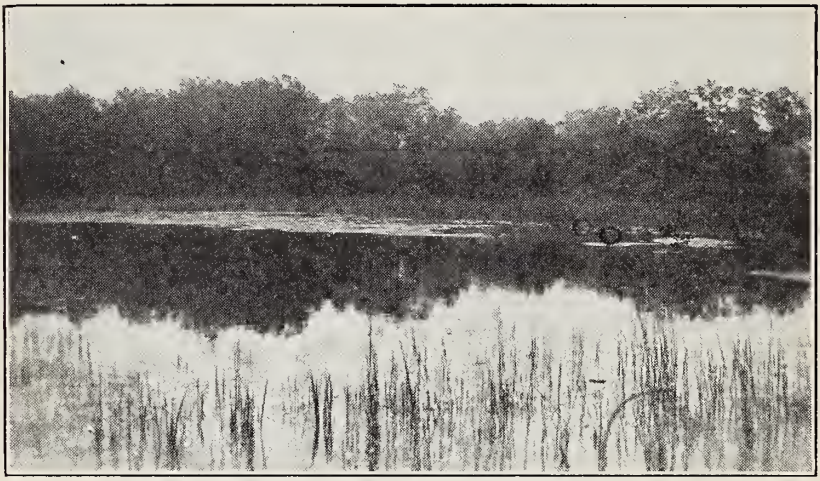


Fig. 48. Where mosquitoes breed by the thousands

time, however, they hang head down from the surface film of the water. They are supported by a tuft of fine hairs at the tail end that floats at the surface. While in this position they are constantly breathing through two little tubes opening to the air at this end of the body. They feed on still smaller living things that inhabit these same waters. (Fig. 49.)

In about ten days the tiny larva has grown considerably larger, as large around as a pin and a quarter inch or more in length. It now changes to a "pupa." This is shaped like a comma. It floats head up and the breathing tubes are on the

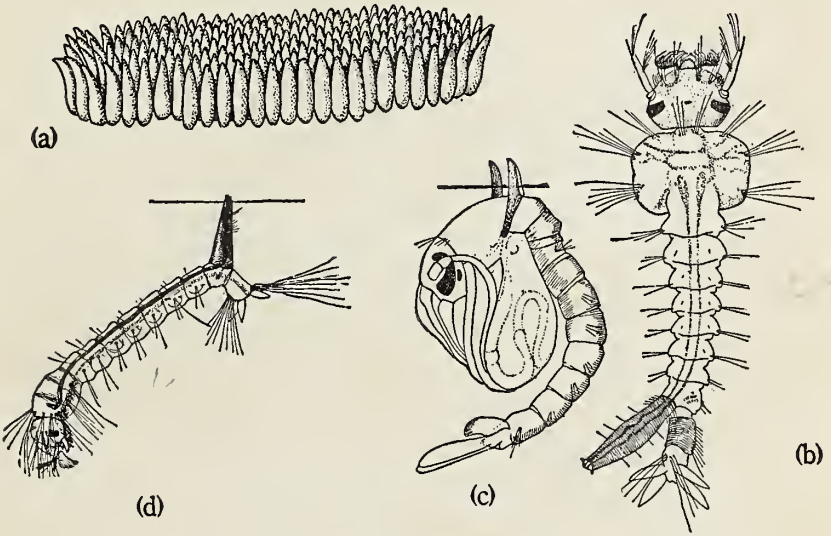


Fig. 49. Life history of the mosquito, *genus Culex*

(*a*) The female lays the sticky eggs that hang together in a raft floating on the surface of the pond.

(*b*) The tiny larvae or "wigglers" hatch from the egg and grow large. The figure is of a nearly full grown larva.

(*c*) The larva changes to a pupa that must stick its breathing tubes above the surface of the water as shown to get air.

(*d*) The position of the larva when at rest obtaining air through its breathing tube.

top of its big head. Of course, neither larvae nor pupae can live unless they can keep on breathing. Oil sprayed on the surface of the water where the mosquitoes breed forms so tough a film that these tiny animals cannot stick their air tubes through it and so must die (Fig. 50). Many small animals,

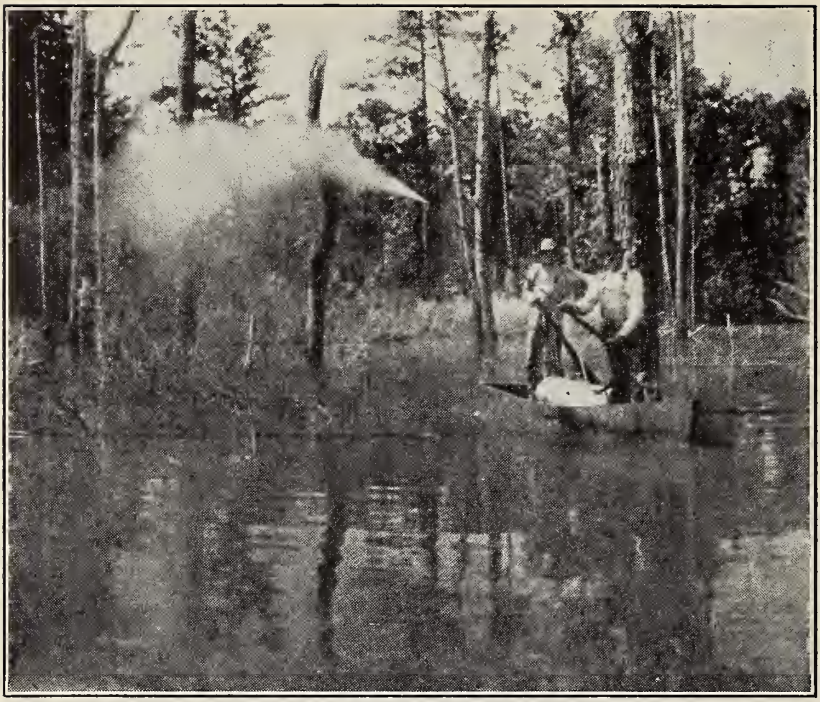


Fig. 50(*a*)

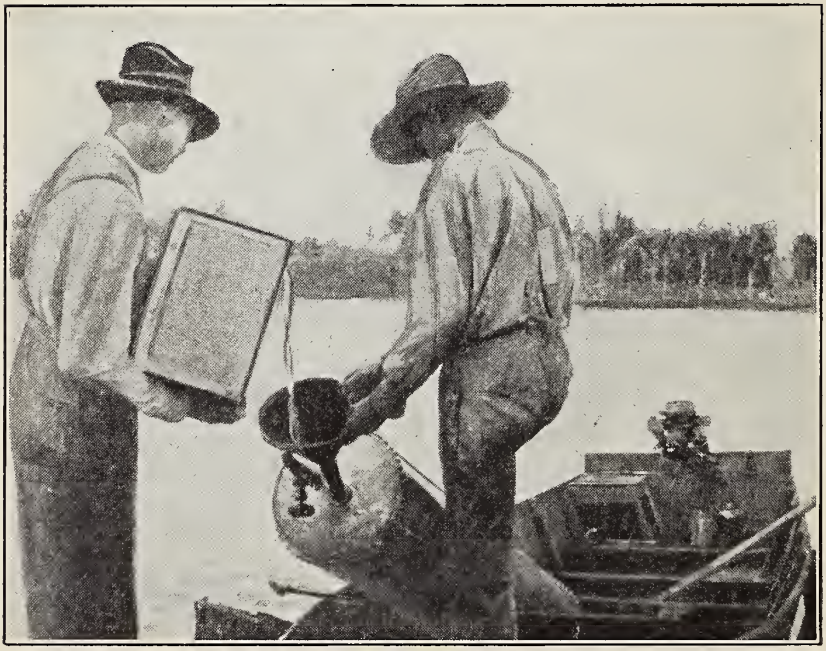


Fig. 50(*b*)

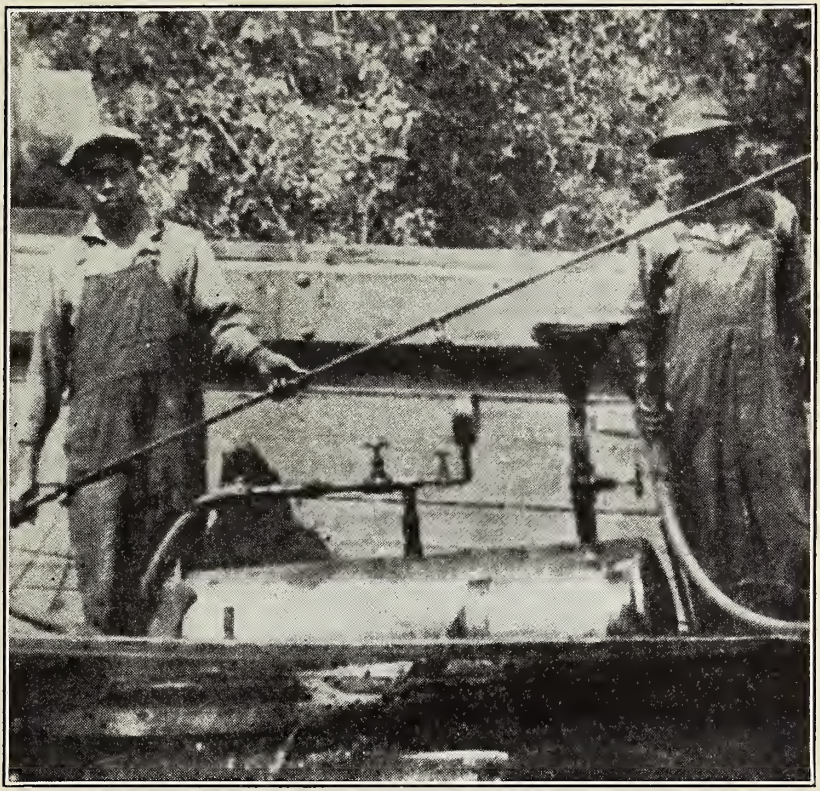


Fig. 50(*c*)

Fig. 50(*a*). Spraying oil to kill mosquito larvae showing the apparatus putting out misty oil-spray which is carried hundreds of feet by prevailing breeze [Courtesy U. S. Surgeon-General]

Fig. 50(*b*). Tank being partially filled with kerosene after which it is pumped full of air under pressure [Courtesy U. S. Surgeon-General]

*Fig.*50(*c*). View of pressure tank and attachments

particularly fish (Fig. 51), are fond of the mosquito larvae and pupae for food. Such animals introduced into ponds and streams help prevent mosquitoes from breeding successfully. (Demonstration 14, Appendix p. 303.)

Finally the skin on the back of the head of the pupa breaks and out of the hole the adult mosquito crawls. It stands on the floating pupal skin to stretch and dry its wings and harden its body. Then it flies away to feed, in time to mate, and, if a female, to lay more eggs.

Today there are probably more people in the world sick with malaria than with any other one disease. The numbers run way up into the millions. It is a very widespread disease, found wherever the Anopheles mosquito lives and that is throughout the tropics and well north and south in the temperate zones. The one drug that cures it is quinine and the world uses more than 600 tons of this every year. Still there are millions of people who suffer from the disease because they are too poor to buy quinine, or too ignorant to know its use. Thanks to the knowledge that the disease can be carried only by the mosquito, malaria is fast disappearing in civilized countries where the breeding places of these pests are drained or rendered uninhabitable for their young by the use of oil sprays.

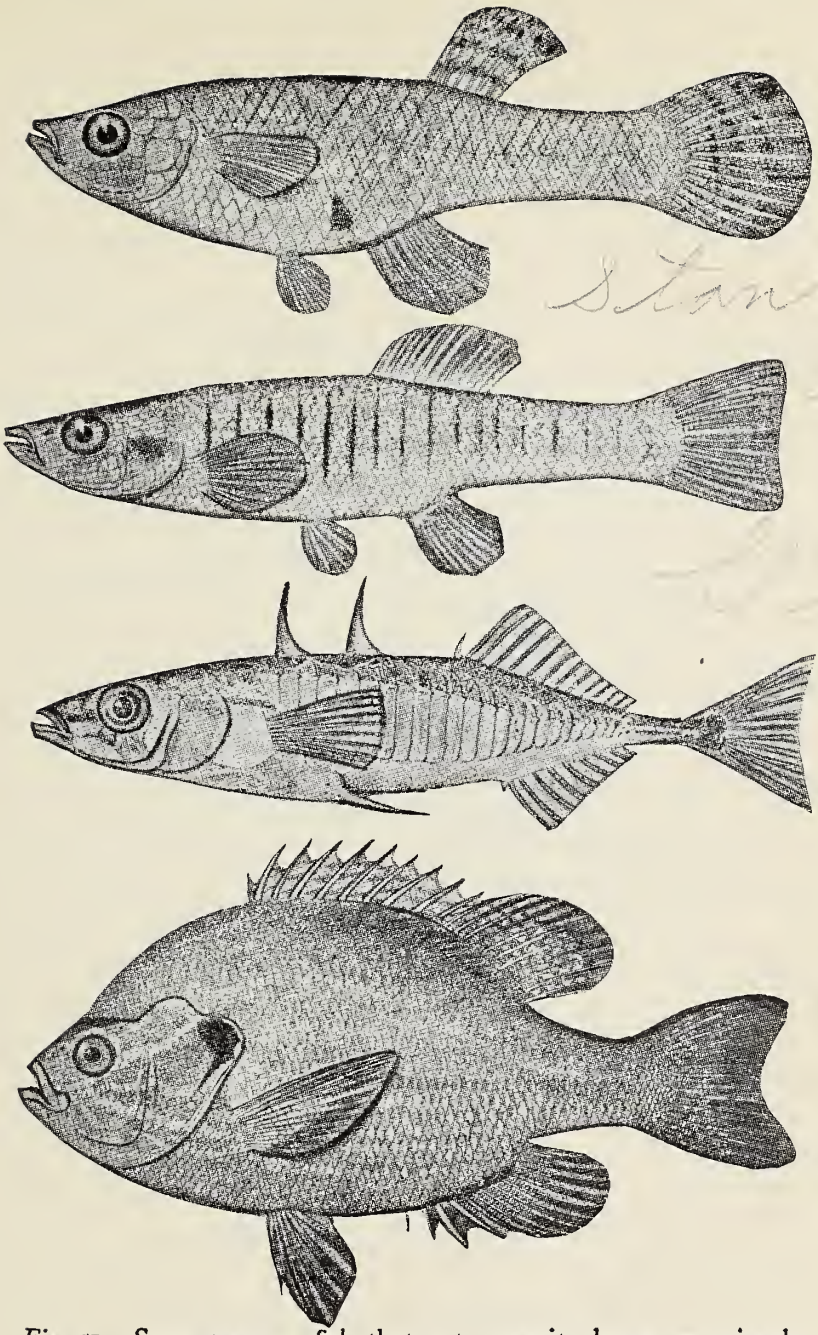


Fig. 51. Some common fish that eat mosquito larvae voraciously

1. Top-minnow, Gambusia affinis (2.5 inches). 2. Killifish, Fundulus Diaphanus (4 inches). 3. Stickleback, Gasterosteus aculeatus (4 inches). 4. Sunfish, Eupomotis gibbosus (6 inches) [Courtesy International Health Board]

YELLOW FEVER AND MOSQUITOES

YET a third time the mosquito was charged with the spread of a murderous disease. This time the guilty fellow was known as *Stegomyia:* the disease in this case was yellow fever. The charge has been amply proved and the guilty culprit ordered killed as rapidly as possible. No more dramatic incidents in the history of the conquest of human ills have been enacted than these in the effort to exterminate yellow fever. No better example can be given of how scientists think their way with nice exactness through such intricate problems.

At the close of the Spanish American War, Cuba, which had been a Spanish possession, was held by the United States. The island population was suffering from many diseases, for Spanish rule had taught the inhabitants little of the art of healthy living. Havana, the chief city, had been for centuries the center from which there spread to neighboring countries deadly epidemics of yellow fever. Immigrants from Havana to New Orleans, Charleston, Philadelphia, and other coast cities, had brought in this dread disease. In 1793 Philadelphia lost one-tenth of its population by yellow fever. The word picture of this outbreak told by one who was himself, at the time, a resident of the city conveys some idea of the horror it produced. Matthew Carey was a member of a committee of citizens to give relief to the sick and to arrange

for hospital care at Bush Hill Hospital. He wrote "A Short Account of the Malignant (deadly) Fever, Lately Prevalent in Philadelphia." In this article printed in Philadelphia on January 16, 1794, he says:

The consternation of the people at this period was carried beyond all bounds. Dismay and affright were visible in the countenance of almost every person. Many homes were scarcely a moment in the day free from the smell of gunpowder, burned tobacco, nitre, sprinkled vinegar, etc. . . . Those who ventured abroad, had handkerchiefs or sponges, impregnated with vinegar or camphor, at their noses or smelling bottles full of thieves' vinegar; others carried pieces of tarred rope in their hands or pockets, or camphor bags tied around their necks. The corpses of the most respectable citizens, even of those who had not died of the epidemic, were carried to the grave on the shaft of a chair, the horse driven by a negro, unattended by a friend or relative, and without any sort of ceremony. People hastily shifted their course at the sight of a hearse coming toward them. Many never walked on the footpath, but went into the middle of the streets to avoid being infected by passing by houses wherein people had died. Acquaintances and friends avoided each other in the streets and only signified their regard by a cold nod. The old custom of shaking hands fell into such disuse that many shrunk back with affright at even the offer of the hand. A person with a crape or any appearance of mourning was shunned like a viper. And many valued themselves highly on the skill and address with which they got to the windward of every person they met. Indeed, it is not probable that London, at the last stage of the plague, exhibited stronger marks of terror than were to be seen in Philadelphia from the 24th or 25th of August until pretty late in September.

In 1853, New Orleans lost 8000 by death from yellow fever. In 1878 the disease invaded the entire Atlantic coast as far north as Boston. Cases of yellow fever were always present in Havana, frequently in menacing numbers.

When the United States assumed charge of Cuba, sanitary measures were at once put into effect. Havana, and as far as possible the whole island, was cleaned up; the essential rules of community health were enforced. The general death rate at once declined but not, however, that for yellow fever.

Fig. 52. Major-General LEONARD WOOD

THE YELLOW FEVER COMMISSION

FINALLY a commission of army physicians was sent to Havana in 1900 to study this disease especially. Cuba was still under military control with Major General Leonard Wood (Fig. 52) in charge. The commission consisted of Doctors Walter Reed (Fig. 53), ranking as Major, James Carroll, Jesse Lazear and A. Agramonte (Fig. 54). At first these doctors spent weeks in examining yellow fever patients and all the organs in the bodies of those

who died with the disease trying to find the bacterium of yellow fever. But they could not find it. Of course, these doctors knew that malaria was spread by the mosquito. They knew, too, that some physicians had claimed that yellow fever was also carried by the mosquito.

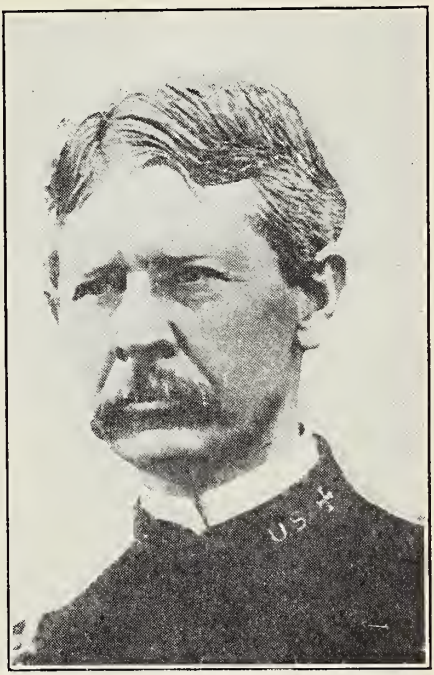


Fig. 53. Major WALTER REED [Courtesy American Museum of Natural History, N. Y.]

Indeed the chief advocate of this idea was Dr. Carlos W. Finley, for many years a practicing physician in Havana. He was a scholarly man, a graduate of the Jefferson Medical College of Philadelphia, a well-beloved physician and a much respected citizen. At a medical congress in Washington in 1881 he had read a paper in which he said that yellow fever was carried from the sick to the well by the mosquito and had even named the Stegomyia mosquito as the carrier. He had tried to prove his point by many experiments but had failed in this completely.

Another man was present in Havana and contributed his bit to the solution of the problem. This was Dr. Henry R. Carter, quarantine officer.

He had been the physician of the Public Health Service in charge of a yellow fever epidemic in a section of Mississippi in 1898. There he had confirmed what before he had suspected that persons do not catch yellow fever until after the patient has been sick for some time. Friends may come to see the sick person for ten days or so and not themselves become sick but after ten days or two weeks they are likely to catch the disease. Moreover he had noted that the patient himself is not essential to the spread of the disease. The quarters in which he has been sick are dangerous. The sick person may be removed or even die, yet two weeks after he first came down with the disease persons going into the house where he was sick are very prone to catch the fever. His house may remain dangerous, a source of infection, for several weeks. Dr. Carter had abundant data to support his ideas for he had observed and recorded the facts over and over in isolated homes where yellow fever had broken out. Dr. Finley and Dr.

Fig. 54. Dr. A. AGRAMONTE [Courtesy American Museum of Natural History, N. Y.]

Carter both discussed their ideas with Dr. Gorgas, Dr. Reed and other members of the Commission. Dr. Carter's facts seemed to support the theory that a mosquito or at least an insect was the carrier of the disease. The Commission decided to test the theory.

These doctors raised the widely prevalent Stegomyia mosquitoes from the eggs. (The name of this mosquito has now been changed to "Aëdes.") They then took these animals in little cages into the hospitals where people were sick and dying of yellow fever. There, in order that each mosquito might become thoroughly infected, it was allowed to bite several patients. It was then kept in its cage for about twelve days, for it was known that it took about that long for a person sick with the disease to give it to a well person. The members of the Commission, Dr. Reed excepted, allowed themselves to be bitten by the mosquitoes. They would not ask any one else to try an experiment so full of peril, until they had shown their own courage. Dr. Reed wanted to try the experiment on himself also but was not permitted to do so for he was in charge of the work and must not risk being sick. Carroll and Lazear both came down with the disease and Lazear, in spite of every care, died. It was necessary to use human beings as subjects in these experiments, for no animal was then known, that suffered from attacks of yellow fever.

Volunteers were called for from the ranks of the Americans in camp near Havana, on whom additional experiments might be tried. William H. Dean of Michigan and John R. Kissinger of Ohio, both privates, and John J. Moran of Ohio, a clerk in the office of General Lee, offered themselves. It was explained to them that they were running a grave risk since half of those who took the disease died. Dr. Reed was authorized to pay men who would submit to the experiment $300.00. But these men refused the money, replying that it was as much their duty to face death by disease, if thereby they could save their fellows, as it was to face the bullets of an enemy.

CAMP LAZEAR

A GROUP of temporary buildings was set up a mile from the main camp to serve the Commission in its experimental work. It was named Camp Lazear in honor of their dead comrade (Fig. 55). The American flag floated over it at half-mast in his honor. One of the houses in the camp was tightly roofed and floored, but the sides were largely left open, covered only with mosquito netting. The doors also were well-screened. The interior was divided by a partition of mosquito-netting. In one of the rooms thus formed mosquitoes that had bitten yellow fever sufferers in the hospital were turned loose. The volunteers lived and slept in this mosquito-infested room and were repeatedly

bitten. In the other room were other volunteers. Only the men bitten by the mosquitoes had the disease; those in the other room, never. The mosquito-net partition made the difference.

It was generally thought up to this time that a person caught the yellow fever by contact with the

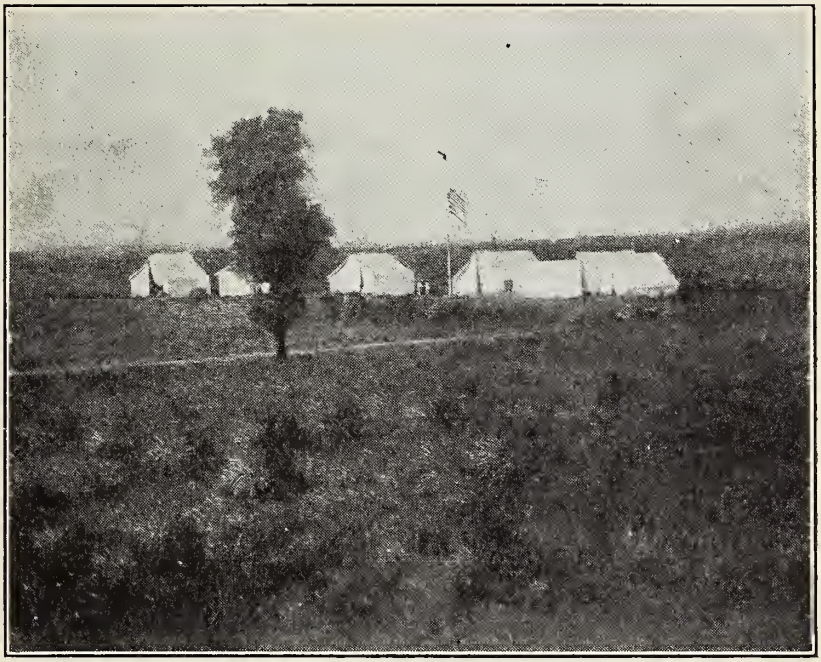


Fig. 55. Camp Lazear [Courtesy American Museum of Natural History, N. Y.]

clothing or bedding of a yellow fever patient. A hut was built at Camp Lazear with small windows. It was poorly ventilated. It was heated by a stove on which a kettle of water was kept boiling so that the air would be warm and moist. Here Dr. R. P. Cooke and several soldiers including Levi E.

Folk, Warren G. Jernegan and James Hildebrand, all volunteers, slept night after night on filthy bedding which had been used by yellow fever patients who had just died in the hospital. The volunteers wore the soiled night shirts of these dead men. It was a nasty job, but none of them took the disease for the hut was screened so that mosquitoes could not get to them. This experiment apparently proved that yellow fever is carried from the sick person to the well person only by one agency, the bite of the mosquito, *Aëdes calopus* (Fig. 46). Further experiments long continued and patiently conducted showed that only in the first three days of the disease can the mosquito be infected from the patient. Then it is twelve days more before its bite can infect another human being.

At once in the spring of 1901 the military authorities acted on the findings of this Commission. The yellow fever patients were put into mosquito-proof hospitals. Doctors were ordered immediately to report to the authorities all cases of yellow fever. The patients were promptly taken to the hospital and their houses and also neighboring houses were fumigated so as to kill all mosquitoes. Cisterns, rain-water barrels and pools were screened so mosquitoes could not breed in them. Stagnant water in swamps, ditches and other places was drained away wherever possible. If this was not possible, the surface of the water was covered

with a film of oil so that the mosquitoes could not breed in it.

THE RESULTS

THE results of these measures were apparent very soon. Few cases of yellow fever occurred in Havana in the summer of that year and by September all were gone except an occasional case brought in by ship or from the country districts. It was the first time in 300 years the city had been free from the disease.

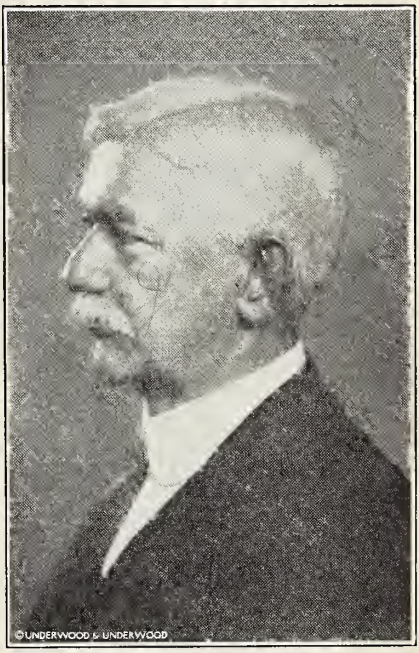


Fig. 56. GEN. WILLIAM C. GORGAS

It was the happy climax of a fortunate combination of circumstances. Here in Havana were Dr. Finley, persistent advocate of the transfer of yellow fever by Stegomyia, Dr. Carter with his data that made the mosquito theory seem plausible, Dr. Reed and his coworkers with their skillful experimental work, and Dr. Gorgas (Fig. 56) with his genius for administration to direct the fight against the disease once the mosquito was proved guilty of carrying the germs.

The world was not slow to learn its lesson. Other countries where yellow fever was prevalent put into effect measures similar to those adopted in Cuba. The Rockefeller Foundation has been especially active in spreading information and providing both workers and funds to drive this old pest off the face of the earth. Success is crowning its efforts. Contrast the two maps showing the areas where yellow fever was prevalent in 1900 and in

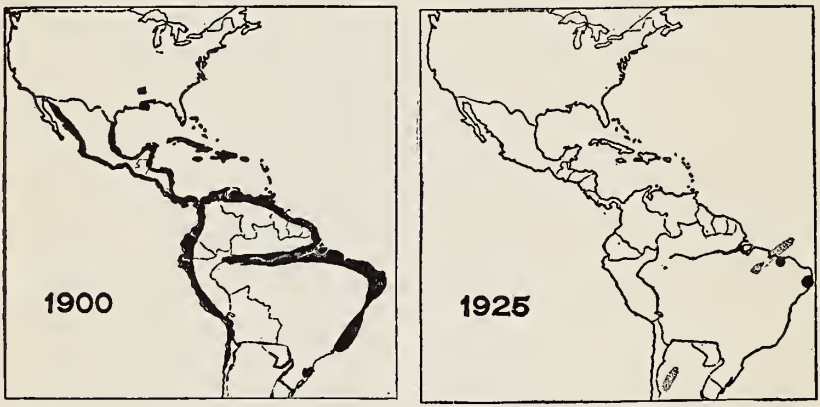


Fig. 57. Where Yellow Fever was found in the Western Hemisphere in 1900 and in 1925 [Report of the Rockefeller Foundation]

1925. (Fig. 57.) Only a single locality in the western hemisphere still harbors the disease. In 1926 only 36 cases of yellow fever were reported in the whole world, three in the western hemisphere. If there are no yellow fever patients, the Aëdes mosquito cannot obtain its deadly charge of infected blood. If there are no Aëdes mosquitoes, the disease cannot be carried from a sick person to a well one. The campaign of extermination conducted

by the Rockefeller Foundation and other health agencies aims to banish both patient and mosquito, so making assurance doubly sure.

Even yet scientists are not all sure that the particular germ of yellow fever has been discovered. Dr. Carroll drew the blood from persons just dead of the disease. He filtered it in a filter so fine it would catch all objects the size of bacteria. But the liquid part of the blood would still give the disease to well persons into whom a little of it was injected. He concluded that the disease must be produced by some organism so tiny it could not be seen under our microscopes. In 1918 Dr. Noguchi, a Japanese scientist working with the Rockefeller Institute, found a tiny spiral bacterium which, in his opinion, is the cause of the disease.

Well may Americans be proud of these heroic doctors and the equally heroic volunteer soldiers and civilians who made these decisive tests possible. We must be proud of the scientific skill of Doctor Reed and his associates. We thrill to know that an American institution, the Rockefeller Foundation, is so successfully trying to drive this disease from the world. We must remember with gratitude the names of these army doctors, soldiers and citizens who risked their lives to find the cause and the cure of the disease. But as Americans we must hang our heads in shame for one thing. When Congress was asked to show the appreciation of the American people for the heroism

of Dr. Lazear, who died in this yellow fever campaign, it granted his widow $5.00 a week, eight years later raising the appropriation to $30.00 per week. The same sum was voted to the widow of Dr. Reed after his death in 1902. Some of the brave soldiers who took part in these experiments suffered want in the later years of the lives which they devoted to our defense. When one recalls how the English people voted Jenner $50,000 for his discovery of a means of preventing smallpox by means of vaccination, a citizen of these rich United States must blush at our niggardly treatment of these heroes of ours.

In addition to those already mentioned the following men heroically submitted to these life-saving experiments in the course of many weeks of experimentation: John H. Andrews, John R. Bullard, A. W. Covington, Thomas M. England, Wallace Forbes, P. Hamann, James F. Hanberry, William Olson, Charles G. Sonntag, Clyde L. West, Edward Weathermarks. At last, in 1928, a bill was introduced in Congress to grant them all more generous pensions, a shamefully belated recognition of their devoted service.

THE PANAMA CANAL

THE lessons learned in Cuba were of great help to the United States when we undertook to dig the Panama Canal. The French people undertook that job once and failed. The mosquitoes drove

them away. The great French engineer, deLesseps, who successfully completed the Suez Canal, was put in charge of the project. Millions of dollars were spent in building great dredges and other machinery for the work. Hundreds of laborers and these machines began work along the proposed line of the canal, in 1881, though not in full force until 1883. Then the mosquitoes began to get in their deadly work. The men sickened and died by the hundreds, of malaria, yellow fever, and other tropical diseases. Other men were sent from France to take their places. The number of sick and dying constantly increased. More money and more men were poured into the enterprise. For eight years the struggle went on, but at last the job was given up. The mosquitoes with their deadly germs were conquerors. The effort had cost 25,000 lives and $260,000,000 in money. The partially completed ditch was bordered with graveyards and abandoned machinery (Fig. 58). Man, the lord of creation, had been defeated by his tiny antagonists.

That knowledge is power was never better shown than when the United States undertook to do what the French canal company had failed in doing. The houses for the men were made mosquito-proof —all were doubly-screened. Along the right of way of the new canal, swamps and marshes were drained or sprayed with oil. In spite of all precautions, there were cases of yellow fever and

malaria, for seven out of ten natives were carriers of the deadly microbes. But the sick were cared for in mosquito-proof hospitals, so they could not be bitten and thus serve as a means of spreading the disease. The cities of Panama and Colon on the Gulf and Pacific ends of the canal were paved, provided with sewers, and furnished with modern

Fig. 58. Abandoned French dredge at Panama Canal, with stagnant water in the foreground [Courtesy Panama Canal Executive Department]

systems to supply pure water. Pure drinking water was furnished the workmen all along the construction line to reduce the danger from typhoid and dysentery. Rats were exterminated to rout bubonic plague.

These sanitary measures cost $20,000,000, but it was money well spent. It made possible the

successful completion of the project (Fig. 59). The United States began digging the canal in 1907 and completed it early in 1914. In eight years the French had removed 108 million cubic yards of earth along the right of way at a cost of 260 million dollars in money. In seven years the United States removed 375 million cubic yards and did all the necessary construction to complete the

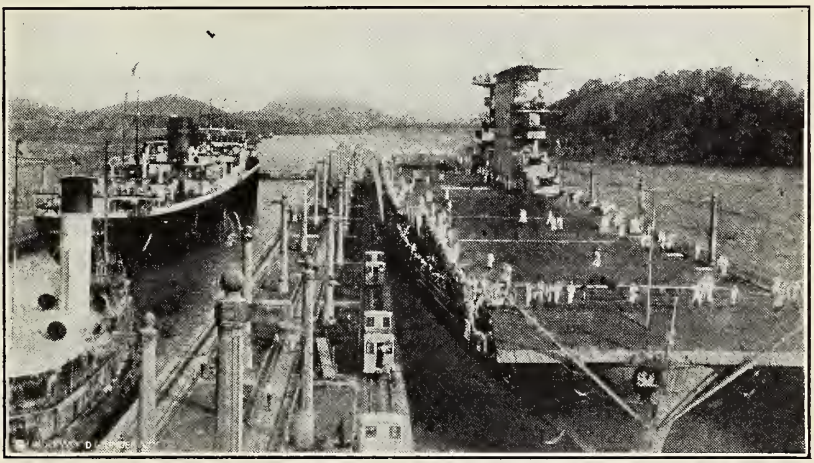


Fig. 59. The completed Panama Canal

canal at a cost of 240 million dollars. Some 60 millions also were spent to acquire the canal zone and to buy the equipment and rights of the old French company. The French had on an average 10,000 men at work during the several years of construction and in that time lost 22,000 by death. The United States had an average of 39,000 men, and lost 4000. W. C. Gorgas, made in after years Surgeon General of the United States Army, was

in charge of the health of the men. To his efficient oversight and to the devotion of the medical men who assisted him, this contrast in mortality is largely due. (Fig. 56.)

THE BUBONIC PLAGUE OR BLACK DEATH

In most civilized countries we now hear very little about the bubonic plague. But for thousands of years this "plague" or "black death" was the most dreaded disease in the world. The sufferer has swelling or "buboes," especially in the arm pits; hence the name. In 1348 half the population of the British Isles died of it. It repeatedly killed off hundreds of thousands of people in China and India. Epidemics of it reappeared in Europe frequently. In 1665, 100,000 people died of it in London alone. Samuel Pepys was then living there and in his diary which has made him famous he tells us what that visitation was like:

June 7th, 1664. This day, much against my will, I did in Drury Lane see two or three houses marked with a red cross upon the doors, and "Lord, have mercy upon us!" writ there; which was a sad sight to me, being the first of the kind that, to my remembrance, I ever saw.

June 15th. The town grows very sickly, and people do be afraid of it: there dying this last week of the plague 112, from 43 the week before; whereof but one in Fenchurch Streete, and one in Broad Streete, by the Treasurer's office.

June 29th. This end of the town every day grows very bad of the plague. The Mortality Bill is come to 267; which is about ninety more than the last.

July 13th. Above 700 died of the plague this week.

July 26th. Sad News of the death of so many in the parish of the plague, forty last night. The bell always going.

July 27th. At home met the weekly Bill, where above 100 encreased in the Bill; and of them in all, about 1700 of the plague.

August 3rd. Rode very finely to Dagenhams; all the way, people, citizens, walking to and fro, enquire how the plague is in the City this week by the Bill; which, by chance, at Greenwich, I had heard was 2020 of the plague.

August 10th. In great trouble to see the Bill this week rise so high, to above 4000 in all and of them above 3000 of the plague.

August 15th. It was dark before I could get home and so land at the Church-yard stairs, where to my great trouble, I met a dead corps of the plague, in the narrow alley, just bringing down a little pair of stairs. But I thank God I was not much disturbed at it. However, I shall beware of being late abroad again.

August 16th. To the Exchange, where I have not been a great while. But Lord! How sad a sight it is to see the streets empty of people and very few upon the "Change." Jealous of every door that one sees shut up, lest it should be the plague; and about us two shops in three, if not more, generally shut up.

August 22nd. I went away, and walked to Greenwich, in my way seeing a coffin with a dead body therein, dead of the plague, lying in an open close belonging to Coome farme, which was carried out last night and the parish have not appointed any body to bury it; but only set a watch there all day and night, that nobody should go thither or come hence; this disease making us more cruel to one another than we are to dogs.

August 28th. To the Exchange, and there was not fifty

people upon it, and but few more like to be, as they told me. I think to take adieu of the London streets.

August 29th. To Greenwich, and called at Sir Theophilus Bidulph's, a sober, discreet man, to discourse of the preventing of the plague in Greenwich and Woolwich and Deptford, where in every place it begins to grow very great.

September 7th. To the Tower and there sent for the Weekly Bill, and find 8252 dead in all, and of them 6978 of the plague; which is a most dreadful number and shows reason to fear that the plague hath got that hold that it will yet continue among us.

September 13th. My meeting dead corpses of the plague, carried to be buried close to me at noon-day through the City in Fenchurch Street. To see a person sick of the sores carried close by me by Grace church in a hacknew-coach. My finding the Angel Tavern, at the lower end of Tower Hill, shut up; and more than that, the alehouse at the Tower Stairs; and more than that, the person was then dying of the plague when I was last there, a little while ago, at night. To hear that poor Payne, my waiter, hath buried a child, and is dying himself. To hear that a labourer I sent but the other day to Dagenhams, to know how they did there, is dead of the plague; and that one of my own watermen, that carried me daily, fell sick as soon as he had landed me on Friday morning last.

September 20th. But, Lord! what a sad time it is to see no boats upon the river; and grass grows all up and down White Hall court, and nobody but poor wretches in the streets! And, which is worst of all, the Duke showed us the number of the plague this week, brought in the last night from the Lord Mayor; that it is encreased about 600 more than the last, which is quite contrary to our hopes and expectations, from the coldness of the late season. For the whole general number is 8297, and of them the plague 7165; which is more in

the whole, by above 50, than the biggest Bill yet; which is very grievous to us all.

RATS AND THE PLAGUE

EVEN in ancient times it was noticed that during a bad spell of the plague dead rats were to be found everywhere, about streets and dwellings and even in country roads and lanes. When numbers of dead rats began to appear, men knew the plague was coming. But no one knew why. When science began to show how one after another of the dread diseases was due to the tiny organisms that infect our bodies, scientists suspected that rats had the plague and in some way gave it to human beings. In 1894, when the plague was raging in Hong Kong, China, a Japanese scientist, Kitasato, discovered the germ that causes it—a tiny egg-shaped bacterium. He grew cultures of it and by injecting the organism, he could give the disease to rats as well as to other animals.

But how did the germ get from the rat to man? It was fairly certain that it did, for men employed to gather up the dead rats and cart them out of the city during the plague were pretty sure to contract the disease. By now scientists were beginning to suspect insects of being disease-carriers wherever an insect might possibly do the job. Rats have fleas and these same fleas often get onto men and bite them. Could they be the carriers? The idea was evidently worth testing.

A rat suffering from the plague was taken in hand, the operator protected by a complete covering of rubber. The fleas were made unconscious by spraying vapor of chloroform onto the rat and were then combed out of its fur. When their body fluids were examined under the microscope (Fig. 60), numbers of the bacteria of the plague were found. A single flea might have hundreds of them. Then well rats were put into one cage and

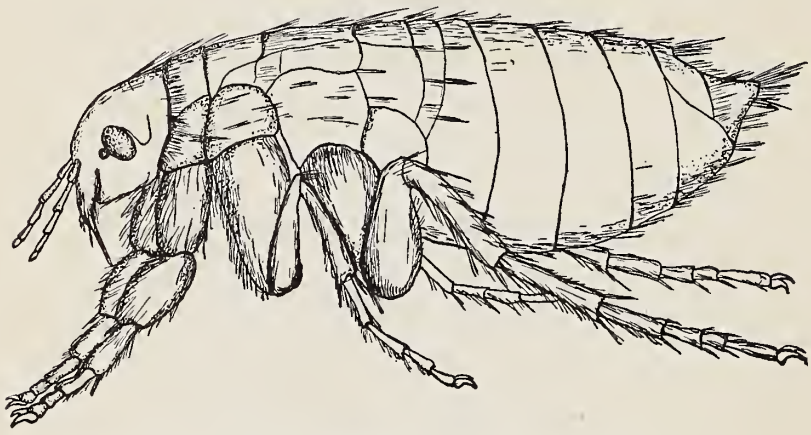


Fig. 60. The human flea, enlarged about thirty times

over this cage was placed a cage of rats sick of the plague. The sick rats died of the plague. Presumably the fleas left them then and hopped down onto the well rats for soon the rats in the lower cage caught the disease. Now the experiment was tried again. But the cage with the well rats was hung above the cage of sick rats at a distance greater than a flea can jump. The sick rats died, but the well rats kept well. A cage of rats sick with the

plague was surrounded by a broad strip of sticky fly paper. A cage of well rats was set beyond this strip. The fleas did leave the sick rats as they died, but they were caught and died on the sticky paper as they tried to reach the well rats. None of the latter contracted the disease. These and similar experiments, often repeated, left little doubt that the fleas transferred the germs of the plague from diseased rats to men.

When bubonic plague appeared in San Francisco in 1907, not only did the city at once try to exterminate the rats, but also other small rodents like chipmunks, squirrels, and ground hogs, for these also are subject to the disease and harbor the fleas. It is no easy job to rid a community of rats. They breed very rapidly. In warm climates they have eight or ten litters of young each year with ten to twenty young in each litter. A pair of old rats may easily produce one to two hundred young in a year. These young grow so fast that they begin to bear young when about three months old. There are probably billions of these worse than worthless pests in the world, destroying crops and produce of all sorts as well as spreading disease.

It was probably some infected rats that escaped from a ship arriving from China that brought the plague to the port of San Francisco. So now ships arriving from countries where the plague is prevalent are anchored off shore and fumigated with gases to kill the rats. Then they are allowed

to come to the wharf to unload (Fig. 61). As many as 2000 dead rats have been found on unloading such a ship.

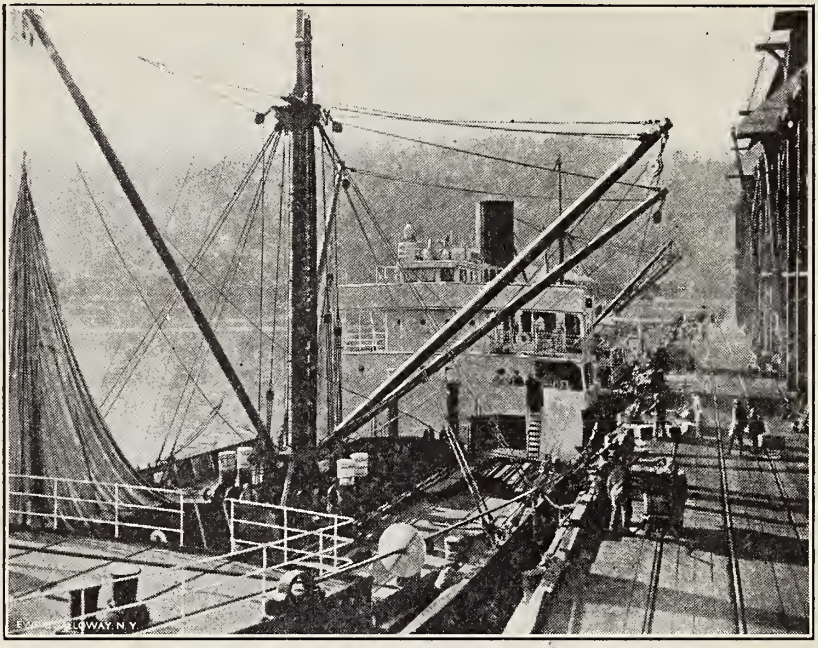


Fig. 61. A ship at a wharf with rat guards on the ropes

CHAPTER V

THE WAR ON DISEASE CONTINUES

TYPHOID FEVER

While Asiatic cholera and the "black death" or bubonic plague were occasionally wiping out thousands, even hundreds of thousands of lives in Europe, when epidemics occurred, there was one other disease that caused even greater loss of life because it was always present. Typhoid fever is almost world-wide in its distribution. It has been, and still is, one of the great scourges of humanity. It was much more deadly among English troops in India than the cholera, although India is one of the worst cholera districts in the world. There were nearly 60,000 cases of it among English soldiers serving in Africa during the Boer War and more than 8000 men died of it there. In our own Civil War more men died of this disease than were killed on the battlefields.

In 1880 Karl Joseph Ebert, then a professor at the University of Zurich, Switzerland, later at Halle, Germany, discovered the bacterium that causes the disease (Fig. 62). Demonstration

15, Appendix p. 304.) Gaffky, one of Koch's asistants, in 1884 grew cultures of this bacterium and by injecting these into guinea pigs produced the disease in them. The germs live in the human intestine and cause sores, especially in certain intestinal glands. These germs produce a high fever in the patient and extreme exhaustion. The

Fig. 62. The bacteria of typhoid fever
[Jordan's General Bacteriology]

intestinal discharges of the patient are loaded with the bacilli. This filth may get into drinking water or milk, or the germs may be carried to food by unclean hands or by flies. (Fig. 63.) So a healthy person may be infected by the use of such polluted water or food. If one recovers from the disease, he is not likely to catch it a second time, but he may harbor the germs in his intestine for many years

and not be sick. Such a "typhoid carrier" may be the source of infection for many new cases and not know that he is a danger to his fellows.

By boiling all drinking water and protecting food from flies and other sources of infection, the Japanese were largely able during their war with Russia (1904) to avoid typhoid fever that in most earlier wars has killed more soldiers than have the enemy's bullets. During the Boer War one English soldier out of every five sent to Africa had typhoid fever. In the Japanese army only one out of every 800 had the disease.

Almoth Wright, a British bacteriologist, devised an anti-typhoid vaccination which was used by the British soldiers during the Great War. He grew cultures, in broth, of the *Bacillus typhosus*, as the germ of this disease is called. These germs were then killed by heat. When the dead germs were injected into the men, they caused the development in the soldier of something that made the man immune to the disease. In the British army on the Western Front only 213 men died of typhoid during the whole war, although this army averaged over a million and a half men all the time. During the first year of the war before the French began to use the vaccine, they lost 10,000 men of typhoid fever.

To tell in detail about all the discoveries made by scientists of the minute creatures that cause disease would fill volumes. Enough has

been told to help one realize how many thousands of lives have been saved through these discoveries, the result of untiring, unselfish work on the part of great men. Pasteur was urged by some of his acquaintances to keep secret his process of treating hydrophobia, so he could make his fortune by it, but he refused. Without thought of gain, these men have given their discoveries to the world so that lives might be saved and human suffering be lessened.

Fig. 63(*a*). Eggs of the fly (*Musca domestica* Linn)

The honor roll of these men has many names upon it, and the list of diseases now largely under control through their efforts is also long. For when Pasteur and Koch

Fig. 63(*b*). Larva or maggot of the fly (*Musca domestica* Linn) [Courtesy American Museum of Natural History, N. Y.]

showed that diseases are caused by bacteria and other tiny living creatures that infest our bodies, they started scientists on a hunt for such germs the world over. Wherever a deadly disease was

prevalent, there the doctors went to discover its cause and the way to prevent it.

Fig. 63(*c*). Pupa of the fly (*Musca domestica* Linn)

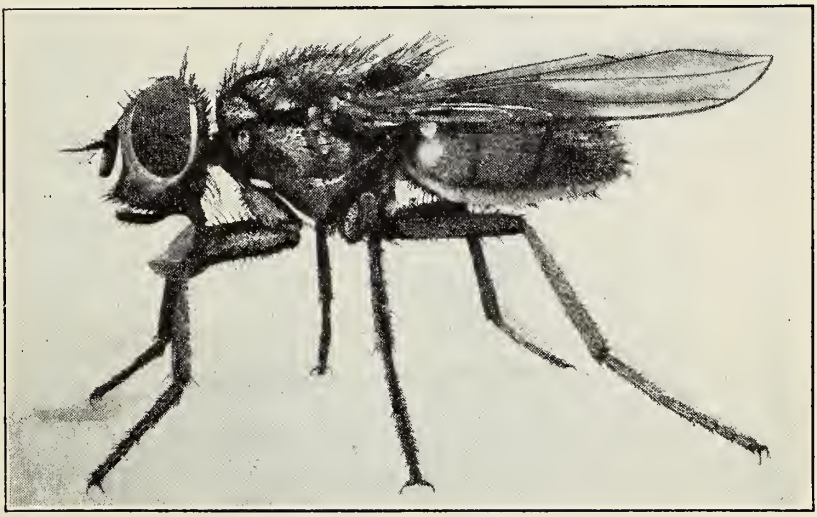


Fig. 63(*d*). Common House or Filth Fly. *a*, *b*, *c*, *d* all enlarged
[Courtesy American Museum of Natural History, N. Y.]

THE GERMS OF TETANUS AND SLEEPING SICKNESS ARE FOUND

In 1890 Kitasato, in Japan, found a means of preventing tetanus or lockjaw. When an animal or a

man gets dirt into a wound which may be only a tiny scratch, this disease may develop. The muscles all over the body including those of the jaws contract rigidly, so the mouth cannot be opened. Kitasato found there was a poison in the blood of the diseased animal that produced these muscular spasms. If he injected the blood of an animal suffering from tetanus into a well animal, the latter promptly had the spasms. But he also found that if he injected a very tiny bit of this poisonous blood into a healthy animal, there developed in its blood an antidote to the poison. He could then inject larger and larger doses of the poisonous blood. The only effect was to produce more and more of the antidote. Then Kitasato and Von Behring, in Germany, found that if some of the blood of this immunized animal was injected into a normal animal, the latter was also immune. The blood of an animal suffering from tetanus did not produce spasms when injected into the normal animal that had been so treated.

The bacteria that produce the poison in the wound were not discovered until 1894 by Nicolaier. These bacteria are found in dirt, especially in soil that has long been cultivated. They remain and multiply in the wound, secreting the poison that gets into the blood and affects the nerve centers in such a way as to produce the spasms. The horse is largely used as the source of anti-tetanus serum. The antidote to the poison is

developed in its blood as described above. If the liquid part of the blood is then injected into a person promptly after he receives a wound, he will not then develop tetanus even if his wound was infected with the germs of the disease. This serum was used constantly during the war, for many men could not help getting dirt in their wounds. It undoubtedly saved thousands of lives.

DAVID BRUCE

In 1895 David Bruce, an English army doctor serving in Africa discovered the cause of *nagana*—a disease fatal to cattle and horses. The little beast that causes the disease lives in the blood of these animals. It is a long, slender, transparent creature, which swims by means of a slender whiplike lash at the forward end of its body. A thin film of living substance runs like the keel of a boat, along its body and part way up the lash. It is not as small as a bacterium, but still so tiny it can only be seen under a good microscope. It belongs to a group of animals known as *trypanosomes* (Fig. 64. Demonstration 16. Appendix p. 304.) Bruce further proved that this creature was carried from a sick animal to a well one by one of a group of flies known as "tsetse flies" (Fig. 65).

This same David Bruce had found, while stationed in the Island of Malta in 1887, a germ which he proved was the cause of a fever prevalent there and elsewhere along the coasts of the Mediterra-

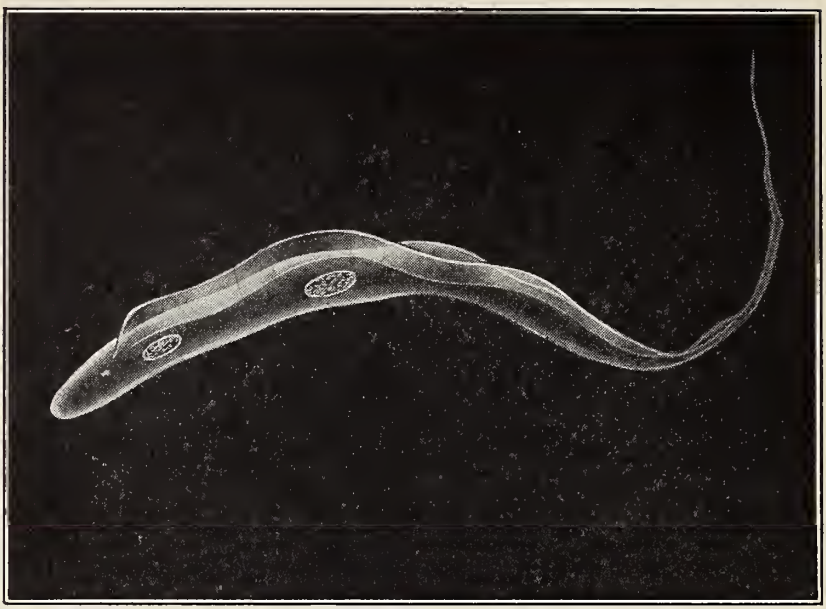


Fig. 64. Trypanosome of Sleeping Sickness. A microscopic parasite with an undulating membrane running down the body. The animal is gelatinous, translucent. This drawing enlarged many hundredfold

Fig. 65. The tsetse fly (*Glossina morsitans*)
[Courtesy of American Museum of Natural History, N. Y.]

nean. It was known as "Malta fever." It was not until 1904 that he had a chance to go back there to find the carrier. Then he proved that the goats from which the inhabitants procure their milk were the guilty animals. The germs did no harm to the goats, but when transferred in their milk to man they produced the fatal disease.

David Bruce was sent to Africa to look for the cause of sleeping sickness and find a cure if possible. The disease was very prevalent among natives and white settlers also, along the shores of Lake Victoria Nyanza. Stricken persons became sleepy, then sleepier, and finally wasted away and died. It was feared in Africa as the cholera or the plague was feared in Europe. Before Bruce arrived, Ford and Castellani had found a trypanosome in patients suffering from the disease which was not found in well persons. Castellani was quite sure that this was the cause of the disease and Bruce also became convinced. But what brought the trypanosome to the well person to make him sick?

Bruce then recalled his work on *nagana* and hunted for a tsetse fly as the carrier. With the help of his wife, who was his very efficient co-worker, he soon found such a fly that was prevalent through the marshy regions along the shores of the lake. They captured some of these flies and let them bite patients who were very sick with sleeping sickness. Afterwards these same flies were allowed to bite

monkeys kept for this purpose. These animals soon came down with the disease. So Mr. and Mrs. Bruce were convinced that the fly was the carrier.

Meanwhile, back in England Dr. Louis Sambon, studying the distribution of sleeping sickness, discovered it was found only in those regions in which the dusky tsetse fly lived. Therefore he concluded that this fly was responsible for carrying the germs of sleeping sickness. He had taken part in the experiments that proved the malaria parasite was carried by the mosquito, so he was wide awake to the relation between insects and disease.

Bruce next found, while examining the blood of all sorts of animals, that the antelopes living in the region harbored this trypanosome in their blood. It apparently did the antelopes no harm, but when a tsetse fly which had bitten them later bit a man, the sleeping sickness was planted in him and he was then marked for death. There seemed but one thing to do. The population must move out of these marshy shores where the dusky tsetse fly lived, up into the highlands back from the lake. This was largely accomplished and sleeping sickness disappeared. It has re-appeared elsewhere in Africa and the battle with tsetse flies still goes on.

Typhus fever is a disease that has repeatedly ravaged European and Asiatic countries. At first it was confused with typhoid fever. During the war it was so terribly prevalent in Serbia and

Russia that over a hundred thousand people died of it in the former country and no one knows how many in the latter. Dr. Louis Sambon prophesied in 1907 that the body louse (Fig. 66) would be found to be the carrier of typhus germs and in 1910 Dr. Nicolle working at the Pasteur Institute in Tunis proved by experiments the truth of this prophecy. In the later years of the war, this same body louse, familiarly known to the soldiers as the "cootie," was proved to be the carrier of the germ of trench fever. The proof of this was obtained through the heroism of men, perfectly well men, who volunteered to let lice which had bitten patients suffering from the disease bite them. These volunteers came down with the disease and several of them died. These unknown soldiers are as worthy of our tributes of gratitude as are those other unknown heroes who gave up their lives in battle. We may doubt the efficacy of war as a means of promoting human justice, but we cannot doubt the efficacy of the war on disease as a means of increasing human happiness.

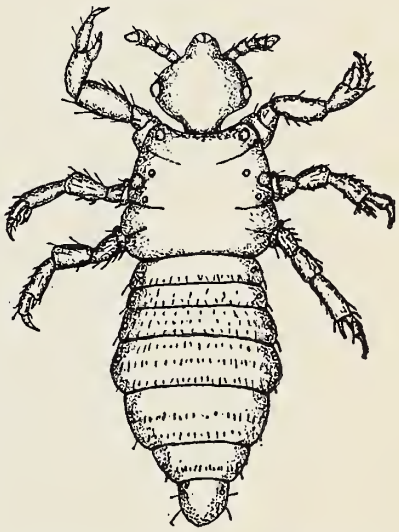


Fig. 66. The body louse (about twenty times its natural size)

THE CAUSE OF HOOKWORM DISEASE IS DISCOVERED

Hookworm disease is a malady that is probably very old, but the cause of it has only recently been discovered. The fact that it is widely prevalent in tropical and adjacent temperate countries and that very large percentages of the population in these regions are infected with the hookworm are also new discoveries. In early times the disease was given a variety of names. In 1838, an Italian physician, Angelo Dubini, discovered the parasite that is the cause of the disease in the Eastern hemisphere and in 1902 Dr. Charles Wardell Stiles described the American species that had been found previously but not recognized as a separate form. The parasite probably was not native to America but was brought in from Africa by slaves, another evil heritage of slavery.

Hookworms live in the intestine of man and certain species are found in similar location in other animals. The worm attaches itself by means of hooked teeth to the lining of the intestine. An infected person may have hundreds of these worms living in the intestinal canal. The adult worm is not very large, only a half inch or so in length and about as large in diameter as a pin (Fig. 67, Demonstration 17, Appendix p. 304). But as they frequently shift their positions the wounds made as they detach themselves (to move to a new site) often bleed freely. This loss of blood, together

with the fact that the worms are absorbing their food at the expense of the patient, causes anemia, a disease in which the person becomes pale, thin, so weak as to be unable to work, and finally dies of this weakness. One of the old names for the disease was "tropical anemia."

The adult worms in the intestine discharge quantities of minute eggs. As these are passed from the intestine of the patient the moist soils in countries that do not have proper toilets may contain hundreds of these eggs. The eggs hatch into larval worms. Careless persons with dirty hands

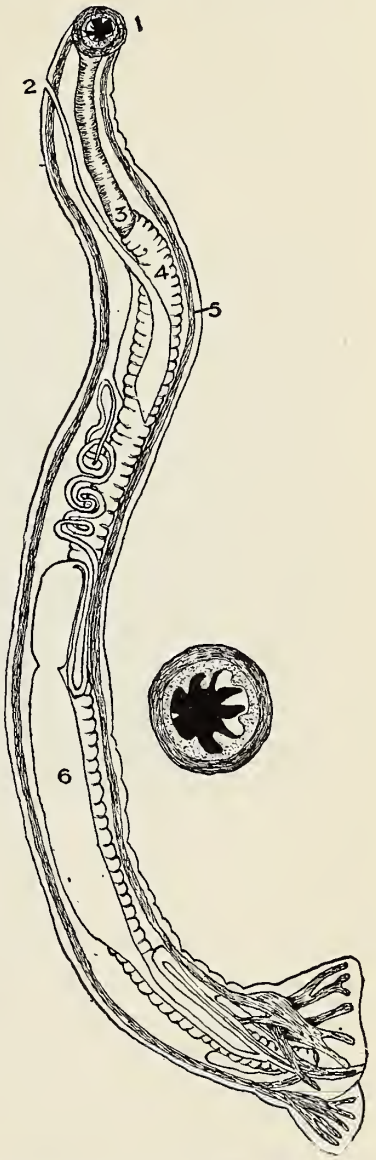


Fig. 67. Diagram of male hookworm (much enlarged). The animal is about .4 of an inch long. This is the common European form (*Anchylostoma duodenale*) introduced into this country by immigrants. The more common species (*Necator Americanus*) probably came in with slaves from Africa. 1. Mouth. 2. Excretory pore. 3. Muscular pharynx by which blood is sucked. 4. Digestive canal. 5. Muscles of body wall. 6. Reproductive organs.
At right is shown mouth of the common American form (also enlarged). Notice the teeth by means of which the animal fastens on to the lining of the intestine and the sharp ridges by which this lining is lacerated to make it bleed.

may infect their food and so allow hookworms to get into their intestines. Much more frequently, however, the worm bores its way (Fig. 68) through the skin of the hands or more commonly through the skin of the feet of barefooted persons who work in places where the soil is infected. The irritation of the skin thus caused has been long known as

Fig. 68. Hookworm larva penetrating the skin, greatly magnified [Report of the Rockefeller Foundation]

"ground itch." When the young worm gains access to the blood stream and has been carried to the lungs, it crawls up the bronchial tubes into the trachea. The trachea and bronchial tubes lead from the throat to the lungs. It is coughed up in the phlegm, and when this is swallowed the worm gets into the intestine. One marvels that a parasite can gain access to the alimentary tract in such

a roundabout way. That it does, however, is demonstrated by the fact that in most cases only persons who go barefooted are found to be infected.

The eggs and the young worms cannot stand low temperatures, and die rather quickly when dried or when exposed to bright sunlight. The disease is therefore confined to the warmer countries and is most prevalent in those regions where the soil is moist, as in coastal plains, river margins or in clay soils in the interior. Agricultural laborers are the persons most commonly infected. The disease is also found among miners and often in mines well north or south of the tropics since mines are usually moderately warm, moist, and dark. Brickmakers also suffer from the disease because they work with moist clay.

When the Saint Gothard tunnel (Fig. 69) was being put through, there was a bad outbreak of hookworm disease among the workmen. This tunnel was cut through the Alps of Eastern France so trains en route to Italy would not have to climb over the high divide. A majority of the workmen were Italians and many of them died of anemia. These men were found to be heavily infected with hookworms. Many of the Italian doctors who went to the workmen's camps at the tunnel thought it was the bad air, the dampness, the inadequate food, and other unsanitary conditions among the workmen that caused their death. But Edouardo Perroncinto, a professor at the University of Turin,

was convinced that the hookworms were the cause of the anemia. He and Bozzolo, another member of the Turin Academy of Medicine, treated the men with drugs to kill the worms and to expel them from the bodies of the sufferers and so cured many cases. This happened in the years 1880 to 1882.

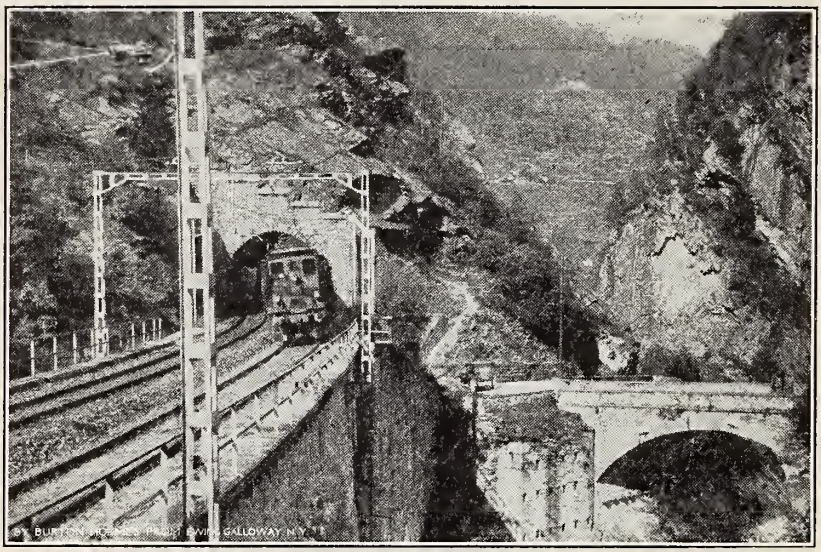


Fig. 69. Train on the St. Gothard Railroad going through the tunnel

In the latter year the great tunnel was completed. The men who had been working in it went elsewhere to get work. Many of them secured jobs in the mines of Italy, France, Germany, Belgium, and England. In this way the hookworm disease was spread, and in these mines where the disease had existed in a limited way, it now broke out in epidemic form. The mine-

owners and the governments as well provided sanitary toilets for the men, they treated the infected men with the proper medicines to kill and expel the worms, new laborers were examined and refused work underground if infected. By such measures the percentage of infected mines was reduced in Germany from 13.1 per cent in 1903 to 0.17 per cent in 1914. In the Liège district in Belgium it was reduced from 22.8 per cent in 1902 to 1.2 per cent in 1913, and in Holland from 25.1 per cent in 1904 to 0.3 per cent in 1913.

THE DISTRIBUTION OF HOOKWORM DISEASE

THE accompanying map shows the regions in which the disease is most prevalent (Fig. 70). It will be seen that hookworm disease is to be found in the southern United States. These regions have been notorious for a long time for the large percentage of anemic, listless people, frequently regarded as lazy. The disease is very prevalent among school children in these regions reducing their inclination and capacity for an education. Various agencies both governmental and private, conspicuously the Rockefeller Foundation, during the present century have been conducting a campaign against the hookworm, not only in southern United States, but the world over. From 1910 to 1925, they examined 1,413,000 persons in the southern states and found 518,668 of these or 36.7 per cent infected with hookworms. During

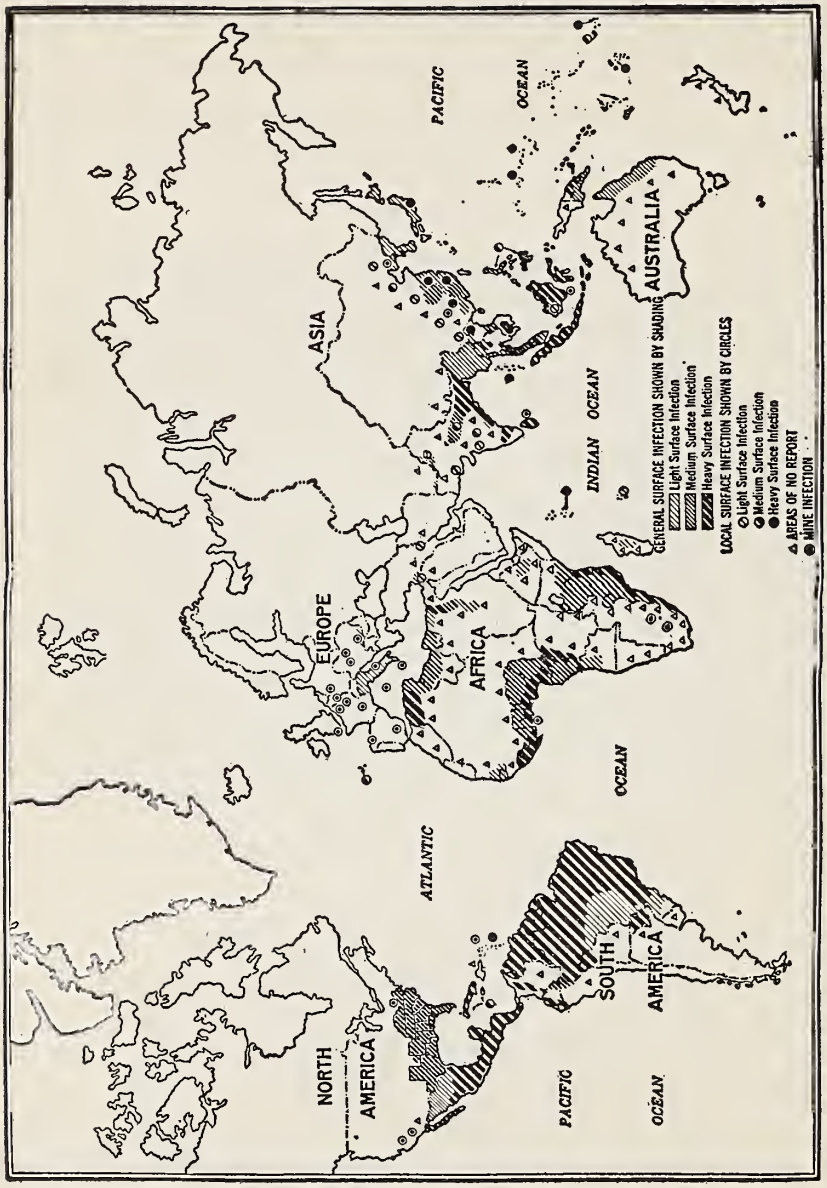


Fig. 70. World-wide distribution of hookworm infection: Surface and Mine
[From Report of the Rockefeller Foundation]

the same years, they have examined throughout the world over 5,000,000 persons and have found over 3,000,000 (59.2 per cent) infected. Their investigations have covered a large number of the tropical and subtropical countries including the West Indies, the countries of Central America, Brazil and Colombia in South America, Australia, Borneo, Ceylon, China, etc., in the East.

Fortunately it is not difficult to recognize the disease and it is farily easy to rid the patient of the parasites. The difficulty is that unless the people of a region can be taught sanitary habits persons become re-infected. It is necessary, therefore, to teach people to use sanitary toilets and that is not an easy matter in many of the worst-infected countries. However, many of the governments in these infected countries are taking vigorous steps to combat the disease and the Rockefeller Foundation is co-operating with them, both by furnishing funds and by providing trained workers. Already in many localities, particularly in the southern United States, there is a decided decrease in the number of infected individuals.

The Rockefeller Foundation has kept data obtained in the original surveys of hookworm conditions in many countries. Then, after treating those persons found infected, they have after several years resurveyed the same countries. The original survey of Ceylon showed that 98.9 per cent of those examined were infected. The resurvey

showed only 52.5 per cent infected. In Costa Rica the original survey showed an infection of 47.6 per cent, the resurvey only 10.6 per cent. In all the countries resurveyed, the original survey showed an average of 87 per cent of the people examined had hookworm. The resurvey showed this reduced to 43.4 per cent. More than that, the average number of hookworms found in infected persons in the resurvey was much less than in the original survey. Thus in Ceylon the average number of worms found in untreated persons was 89. But six years after treatment the average number in infected persons was only 45.

CHAPTER VI

THE EFFECTS OF THESE DISCOVERIES

DIPHTHERIA

Let us see now what the result of these discoveries has been on the number of deaths from these several diseases. These results can best be seen in many cases by noting the death rate in large cities. Cities usually have efficient health officers who are alert to learn every way in which the health of the people can be protected and are quick to adopt new methods. It takes new ideas a long time to reach the remoter parts of the country, but large cities get the news quickly. It would be expected that new discoveries would be promptly put to use in the great cities.

There is given on page 158 a table which shows the death rate from diphtheria in Chicago for sixty years. The figure opposite each year shows the number of persons in every 100,000 of the population who died of diphtheria that year. To illustrate, suppose that in a city with 80,000 people, there were 38 deaths from diphtheria. Since 80,000

DEATH RATE FROM DIPHTHERIA PER 100,000 OF POPULATION IN CHICAGO

Year	Per Cent	Year	Per Cent
1867	79.1	1897	57.9
1868	77.4	1898	43.7
1869	91.8	1899	56.4
1870	139.3	1900	49.5
1871	73.6	1901	29.4
1872	97.2	1902	34.8
1873	62.1	1903	34.4
1874	44.0	1904	21.5
1875	67.2	1905	22.2
1876	184.5	1906	27.7
1877	135.8	1907	27.1
1878	120.2	1908	26.5
1879	196.9	1909	31.7
1880	290.7	1910	37.4
1881	186.9	1911	39.1
1882	134.3	1912	40.9
1883	140.9	1913	31.9
1884	143.7	1914	40.6
1885	152.2	1915	27.7
1886	185.2	1916	31.5
1887	184.9	1917	48.2
1888	161.6	1918	27.7
1889	161.4	1919	22.9
1890	114.7	1920	23.1
1891	118.2	1921	24.5
1892	129.0	1922	20.1
1893	117.1	1923	19.8
1894	107.4	1924	12.6
1895	120.1	1925	8.0
1896	76.2	1926	7.3

is four-fifths of 100,000, this rate of 38 deaths must be increased by one-fourth to make the five-fifths or full 100,000. One-fourth of 38 is 9.5 which

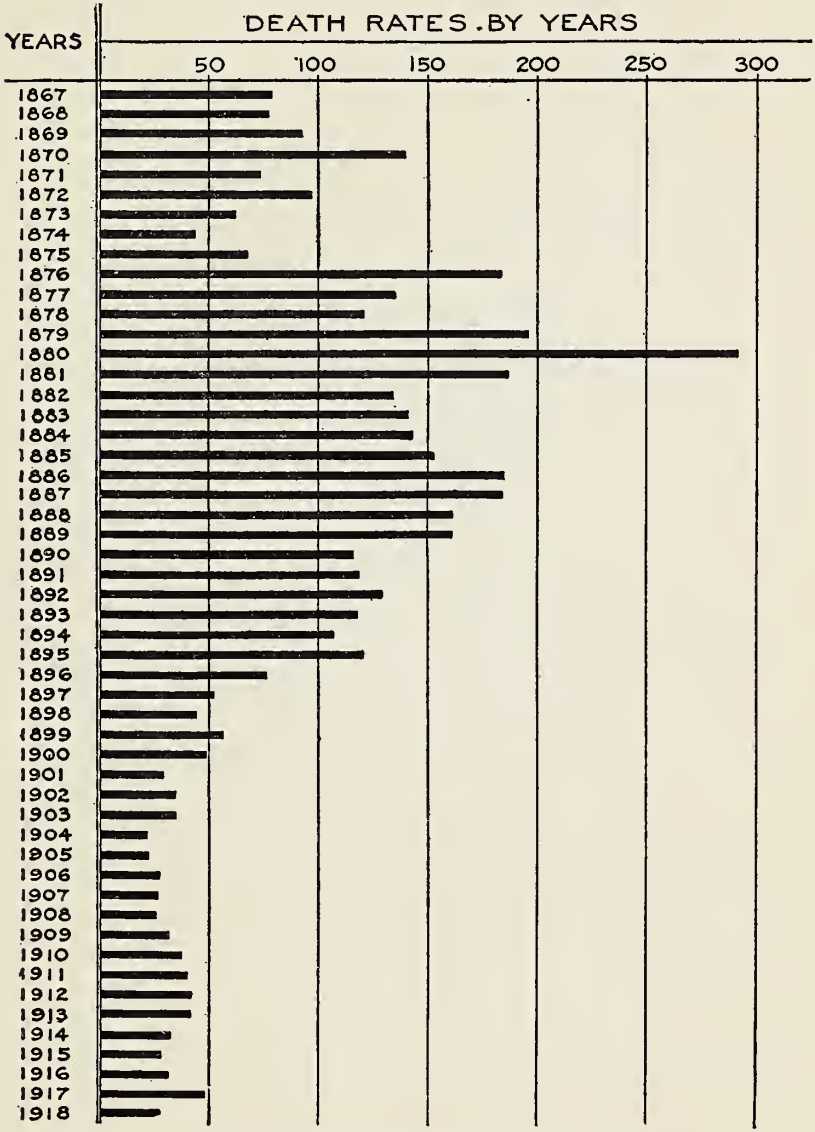


Fig. 71. Death rate from diphtheria per 100,000 population in Chicago for fifty years

added to 38 makes 45.5, the death rate per 100,000 in this city.

Remember in looking over this table that it was in 1883 that Klebs discovered the bacterium that causes diphtheria. In 1888 Yersin and Roux proved that it is the toxin or poison which these bacteria make that kills the patient. Then Von Behring and Kitasato found how to produce antitoxin and how to inject it into animals. In 1891 Von Behring used this antitoxin as a cure for diphtheria in children. Roux obtained the antitoxin in quantity from horses and used it to cure many children of diphtheria in Paris hospitals in 1894. The antitoxin treatment was first given in Chicago in 1896. In 1911 the Schick test was discovered to see if one is susceptible to diphtheria. In 1913 the toxin-antitoxin treatment was devised to make a person develop in his blood so much antitoxin that he will not catch diphtheria. These dates are underscored in the table.

There is given on the preceding page a chart (Fig. 71), that shows these same facts in a way to make the story visible at a glance. The line for each year is drawn in proportion to the number of deaths per 100,000 population. A long line means many deaths, a short line, few. The average death rate for the twenty years, 1876–1895, before antitoxin was used in Chicago, was 154.3. For the twenty years, 1897–1916, after it was introduced, the rate was 41.4. If we again represent

these figures by lines the contrast is shown here.

If the death rate of the first twenty years had continued during the second twenty years, 35,712 more persons would have died of diphtheria than did die

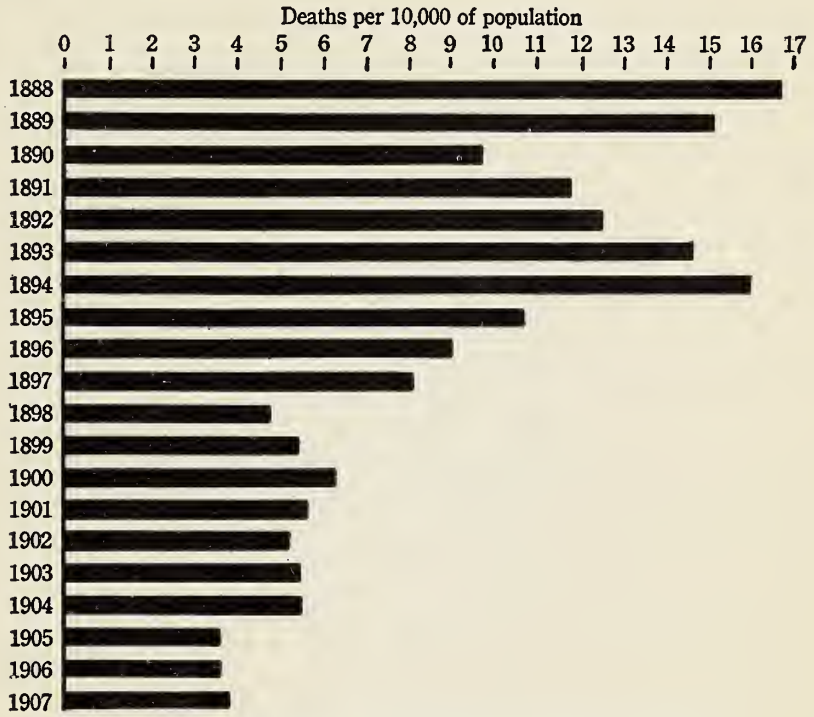


Fig. 72. Twenty years of diphtheria in New York City
The antitoxin treatment was begun late in 1894

of that disease. This is what antitoxin has done just in *one* city.

There is also given a graph (Fig. 72) showing the deaths from diphtheria during twenty years, for every hundred thousand of population in New York City. The antitoxin treatment began to be

used here late in 1894. Note the successive epidemics of the disease. One is just disappearing in 1888 to 1890. Another runs from 1891–1898. The next one, 1899–1906, has a much lower death rate since antitoxin has been available.

In the table below there is given the average death rate per 100,000 population from diphtheria, during five-year periods in several large cities, both in the United States and in foreign countries. Note the drop in the death rate from 1896–1900 as compared with the preceding period, 1891–1895. Remember, it was in 1894 that the antitoxin treatment was given by Roux in Paris to large numbers of children, so demonstrating its efficiency. (The pupils will find it interesting to graph some of these

AVERAGE DEATH RATE FROM DIPHTHERIA PER 100,000 POPULATION FOR A NUMBER OF FIVE-YEAR PERIODS IN SEVERAL CITIES

	Boston	N. Y.	Phila.	London	Paris	Berlin	Stockholm
1881–1885	151	170	139	41	87	172	107
1886–1890	120	162	83	42	70	91	71
1891–1895	120	130	125	58	45	76	95
1896–1900	75	67	94	47	13	31	52
1901–1905	39	52	40	20	17	17	25
1906–1910	28	39	34	14	8	27	17
1911–1915	22.6	25.8	22.6	9.3	('11–'17)		
1916–1920	25.7	20.8	23.3	13.3			

figures as has been done for Chicago and New York.)

For the registration area of the United States the death rate from diphtheria per 100,000 of population was:

1890	97.8	1915	15.7
1900	43.3	1920	15.3
1910	21.4	1924	9.4

In Massachusetts, the averages for five-year periods are as follows:

1875–1879	155	1895–1899	51
1880–1884	107	1900–1904	36
1885–1889	85	1905–1909	23
1890–1894	64	1910–1914	17

The figures for England and Wales were:

1858–1860	37.2	1881–1885	15.6
1861–1865	24.8	1886–1890	17
1866–1870	12.7	1888–1892	37
1871–1875	12.1	1893–1897	61
1876–1880	12.2	1898–1902	35
		1903–1907	15

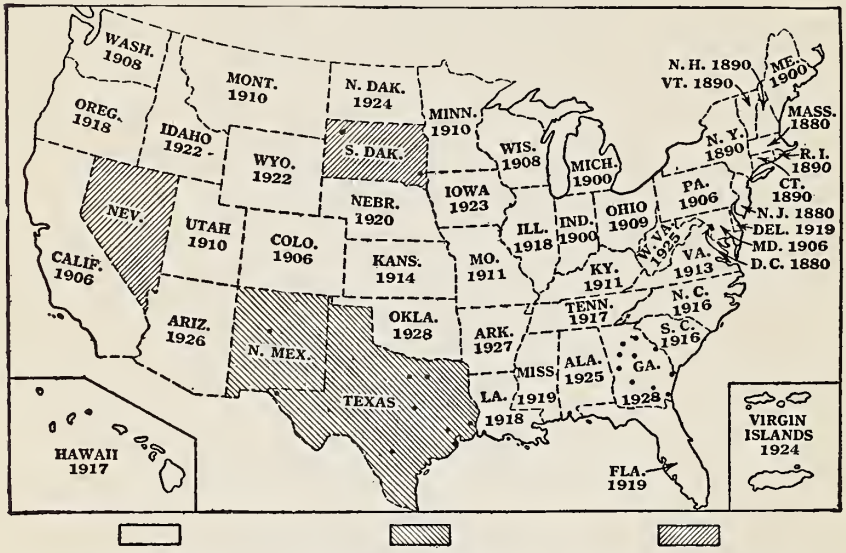
BIRTH AND DEATH REGISTRATION

It is not an easy matter to obtain such facts even for recent years and in many countries quite impossible for earlier dates. In our own country the Census Bureau did not begin to issue its annual reports on deaths and births until 1900, largely because so few states had laws compelling physicians to report on such matters. In 1880 only

Massachusetts, New Jersey, and the District of Columbia were gathering data in regard to the number and causes of deaths. These constituted "the registration area." The accompanying map

Fig. 73. DEATH REGISTRATION AREA: 1928

44 States, the District of Columbia, and 10 registration cities (indicated thus •) in non-registration States; includes 95.4 per cent of total population of the United States.



REGISTRATION STATES
States which have satisfactory registration laws and which actually register at least 90 per cent of all deaths.

LAWS UNDER TRIAL
States with good laws, such as the Model Law, which have not been in force sufficiently long to bring registration up to 90 per cent.

UNSATISFACTORY LAWS
States with laws which do not and can not secure good registration of deaths; for example, laws based on the "county system."

Figures indicate years in which states were added to death registration area.
Department of Commerce, Bureau of the Census, Division of Vital Statistics, Washington.

(Fig. 73) shows the years in which other states adopted adequate laws requiring reports on the causes of death. By 1900 the New England States, New York, New Jersey, Michigan, and the District of Columbia were all in the registra-

tion area. This area has since been steadily increasing, but there are still some states which do not report on these matters. We are, therefore, not sure in the United States what are the chief causes of death. Such neglect is quite inexcusable in a country as progressive as ours.

The first attempt to gather facts in regard to the number of deaths and their causes was made in London in 1532. Weekly "bills of mortality" were issued by parish clerks of the Church of England. Each year a summary of these was printed in a yearly bill of mortality. In the time of King Henry VIII this practice had spread throughout England. These weekly bills covered only the members of the Church of England, not the entire population. Still the facts given are valuable, for they show what diseases were prevalent and which ones were most fatal. They are difficult to interpret, however, for the names of diseases have changed much since then. Thus in a yearly bill of 1668 green-sickness is given as the cause of the death of one person, King's evil (epilepsy) of 39, Rising of the Lights caused the death of 175, and Griping in the Guts of 2415. It was not until 1837 that civil registration took the place of this registration by parish clerks.

France began the official registration of marriages, births, and deaths in 1539. Sweden started a system of parish registration of similar facts in 1608. In 1666 Canada took the first public census.

Norway began to register vital statistics nationally in 1735, Sweden followed in 1748. The first census by the government in this country was taken in 1790. It was merely a count of the number of people living here. Most European countries similarly took a census of their population in the early years of the nineteenth century. But, it was not until 1902 that the Census Bureau was established in this country. It is, therefore, evident, that until recently, then only in a few countries, has it been possible to find the data showing the effects of the discoveries of the causes of disease.

Long before the nature of the real cause of disease was known the death rate was steadily falling in most civilized countries. This was true because the living conditions were improving; people were keeping themselves and their homes cleaner; they were living in less crowded quarters, eating more wholesome foods. Doctors were becoming more skillful, hospitals more sanitary; cities were providing better sewage systems and better water supplies. This general improvement accounts in part for the decline in the death rate even in such diseases as diphtheria. On page 170 is a table showing the crude death rate per 1000 population in several foreign countries and in the United States for five-year periods from 1881–1920. (From U. S. Mortality Report, 1923.) There are some blanks since the complete

data are not available for some countries. A graph is made (Fig. 74) showing the death rate for three countries. Note that the lines run down from 1881 to 1922, except for the 1916–1920

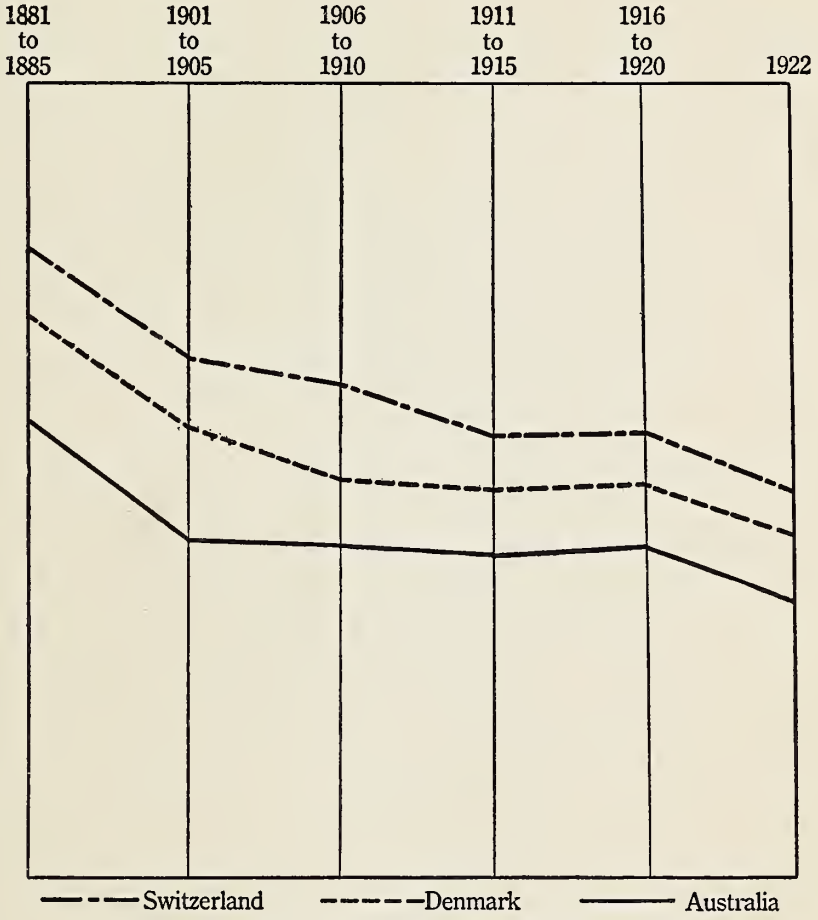


Fig. 74. Graph of declining death rate from all causes

period. In 1918 there was a bad epidemic of influenza the world over, which increased the death rate greatly. It is suggested that the pupil fill in the additional lines. The graph is drawn to

scale so that two inches in height above the base line represents a death rate of 15.

RESULTS OF THE WAR ON TYPHOID FEVER

Let us look now at the data for typhoid fever. Keep in mind that although in 1880 Ebert discovered the bacterium which causes the disease, this was not proved the cause by Gaffky until 1884. The bacterium is found in large numbers in the discharges from the intestine of typhoid patients. When such filth gets into drinking water, or milk, or onto food that is eaten, the germs are transferred from the sick to the well person.

POLLUTED WATER A SOURCE OF TYPHOID FEVER

As an illustration of an epidemic of typhoid fever in which the germs were carried in polluted water, we may take the case of Lowell and Lawrence, Massachusetts. Both of these cities are located on the Merrimac River. Lawrence is about nine miles downstream from Lowell. In 1890 and 1891 an epidemic of typhoid fever broke out in Lowell. That city was supplied with water drawn from the river and used without any treatment. The epidemic was traced to a number of cases that occurred in North Chelmsford, a suburb of Lowell. A small stream, Stony Brook, which runs through this suburb was overhung by a number of privies. When several cases of typhoid occurred in North

Chelmsford, the brook became polluted by intestinal discharges and the bacteria were carried down to the Merrimac River. As a result, 132 persons died in Lowell from typhoid fever. Lawrence also had an outbreak which caused 74 deaths in that city. The climax of the epidemic came in Lawrence about two months after the climax in Lowell.

Both these cities were subject to another epidemic, though not so severe, in 1892. Stimulated by the evident danger of using Merrimac River water without treatment, Lowell began boring deep wells from which to obtain its water supply. Lawrence, on the other hand, put in a filtration plant to purify the river water. There are given below two graphs (Figs. 75, 76) which show the results of these improvements. It will be noted that in each case the death rate from typhoid fever dropped very promptly after the completion of these projects.

MILK, A CARRIER OF TYPHOID GERMS

MILK is often a carrier of the bacteria that cause typhoid fever. Since it is a good food for them they increase rapidly in it, especially if it is not kept cold. The United States Hygienic Laboratory of the Public Health and Marine Hospital Service gives, in its Bulletin No. 56, the facts about hundreds of cases of typhoid fever epidemics that have been traced to milk.

ANNUAL CRUDE DEATH RATE PER 1000 POPULATION

Country	1922	1921	1916 to 1920	1911 to 1915	1906 to 1910	1901 to 1905	1881 to 1885
United States (registration area, exclusive of Hawaii)	11.8	11.6	14.5	13.9	15.1	16.2	
Australia	9.2	9.9	10.8	10.7	10.7	11.7	15.7
Austria	17.3	16.9	21.9	20.0	22.3	24.2	30.1
Belgium	....	13.8	15.8	14.6	15.9	17.0	20.6
Chile	28.4	32.7	30.6	28.9	31.3	30.2	26.9
Denmark	11.9	11.0	13.1	12.8	13.7	14.8	18.4
England and Wales	12.8	12.1	14.5	14.3	14.7	16.0	19.4
Finland	14.4	14.0	19.5	16.1	17.4	18.6	22.2
France	17.5	17.7	20.2	19.3	19.2	19.6	22.2
Germany	....	13.9	19.0	17.7	17.5	19.9	25.3
Ireland	14.1	14.2	16.6	16.8	17.3	17.6	18.0
Italy	....	16.1	21.8	19.3	21.0	21.9	27.3
Japan	22.3	22.7	23.6	20.0	20.9	20.5	
Netherlands	11.4	11.1	13.7	12.8	14.3	16.0	21.4
New Zealand	8.8	8.7	10.7	9.2	9.8	9.9	10.9
Norway	11.9	11.5	14.1	13.2	13.8	14.5	17.2
Ontario, Province of	11.4	11.8	13.2	12.3	14.0	13.0	11.4
Scotland	14.9	13.6	15.0	15.7	16.1	17.0	19.6
Spain	....	21.1	24.6	22.1	24.0	25.9	32.6
Sweden	12.8	12.4	14.6	14.0	14.3	15.5	17.5
Switzerland	12.9	12.7	14.9	14.3	16.0	17.5	21.3

In Newark, N. J., between October 15 and November 23, 1906, there were 103 cases of typhoid, 69 of which were on the route of one

dairy. There was at this dairy farm one person who, although ill with typhoid, continued to milk

Fig. 75. Twenty years of typhoid fever in Lowell, Mass., showing deaths per 100,000 population. Several deep wells were bored during 1893–1895 and the water was taken from them instead of from the Merrimac River

the cows until October 8th, when he was too sick to do so longer.

In January, 1902, an outbreak of typhoid fever occurred at Niles, Ohio. During the first three

months of this year there were 107 cases, 63 on the route of one milkman. A boy on this farm was found to be sick with typhoid.

Elkton, Maryland, a little town of 2542 people, in 1900 had a typhoid fever epidemic when the disease broke out in 32 families in one month.

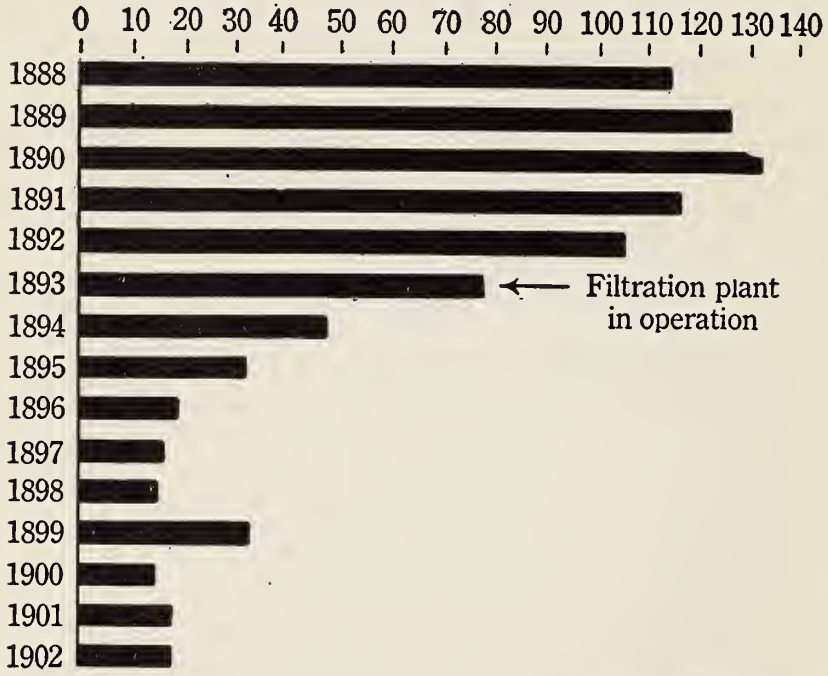


Fig. 76. Fifteen years of Typhoid Fever in Lawrence, Mass., showing deaths per 100,000 population. Filtration plant put into operation about the middle of 1893

These all bought milk from one farmer. Two sons who had helped milk, and their mother who handled the milk, all had typhoid.

In Palo Alto, California, in 1903, 232 persons were taken ill with typhoid out of 900 supplied with milk from one dairy. At this dairy the milk

cans were washed with water from a creek that received the drainage from several houses where there were cases of tyhpoid fever.

Not only does milk carry the germs of typhoid fever, but of many other diseases as well. It is very necessary, therefore, that persons who milk cows and handle milk be healthy and cleanly and that milk containers be carefully sterilized. As a further safeguard milk is often pasteurized (see p. 28) to kill the disease germs it may contain.

Now examine the graph given to show the history of typhoid fever in Chicago (Fig. 77). Chicago is on the shore of Lake Michigan. In the early days, the sewage was dumped into the lake and the drinking water was taken from the lake. Recently the sewage is largely emptied into drainage canals that carry it to the Illinois River. The water is treated with chlorine to kill any disease germs that may, in times of heavy rains, wash into the lake. Note, too, the decrease in the death rate from typhoid fever as the milk supply has been partly, then entirely, pasteurized.

VACCINATION AS A PREVENTATIVE OF SMALLPOX

SMALLPOX is a very old disease. It was known in India several hundred years B.C. Brahman priests as early as 300 B.C. used to rub the liquid discharged from a broken pustule on a person suffering from a mild attack of the disease onto the skin of a

well person so as to give that person the disease. It was hoped he would have a very mild case, too, and so escape the disease in its deadly form when

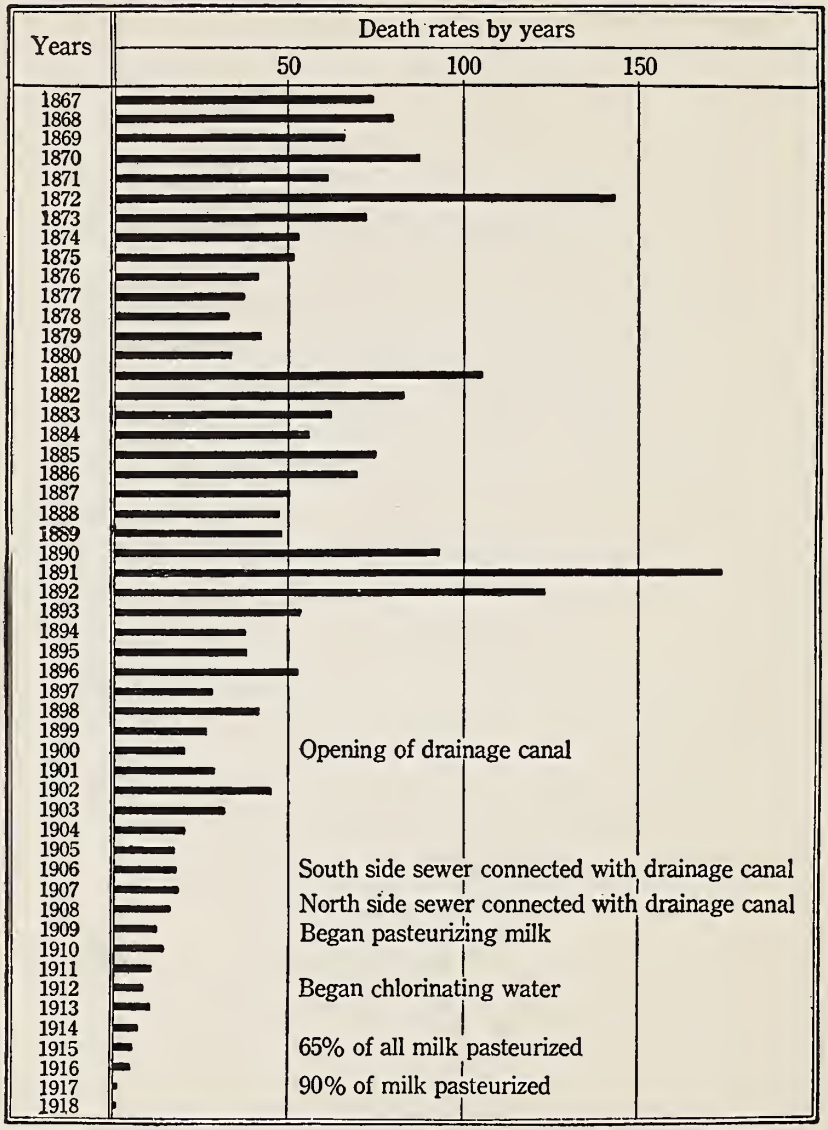


Fig. 77. Typhoid Fever in Chicago. Deaths per 100,000 of population, Chicago Health Department Report, 1911–1918.

there was an epidemic. The disease is still very prevalent in India. In the period 1918–1923, there were 663,553 deaths from smallpox in that country.

The disease spread from India into China, thence to Western Asia, to Turkey and so to Europe. The first recorded case of smallpox occurred in London in 1628, although the disease had been known in England for a hundred years before that. The method of inoculation described above as practiced in India was also used in England. This was not a very good method of prevention since sometimes the person inoculated had smallpox severely and died, sometimes other diseases worse than smallpox were thus transferred from person to person. Jenner's discovery of vaccination was a safe and sure method of smallpox prevention. This, you will remember, was announced in 1788, though it was not widely adopted even in England for several decades after that.

In Europe, during the eighteenth century, there were in round numbers 60,000,000 deaths from smallpox. In the five-year period, 1781–1785, there were reported on an average 18,880.6 deaths, of which 1888.9 or 10% of the total were due to smallpox. The following table puts these and similar data for several succeeding periods into form which makes it very evident that the decrease in smallpox was due largely to vaccination.

	Average Number of Deaths per Year	Average Due to Smallpox	Per Cent of Total Deaths Due to Smallpox
1781–1785	18,880.6	1,888.9	10.0
1786–1790	19,657.4	1,684.6	8.6
1791–1795	20,228.4	1,730.0	8.5
1796–1800	19,131.8	1,965.4	10.3
1801–1805	18,568.8	1,309.8	7.0
1806–1810	18,577.8	1,197.0	6.4
1811–1815	18,300.6	859.8	4.7
1816–1820	19,853.0	711.8	3.6

(The data for these figures are calculated from the statistics given in Creighton's "History of Epidemics in Britain.")

In the 20-year period, 1660–79, there were 417 deaths from smallpox in London per 100,000 population. The following figures show the decline in the rate in successive periods. (See graph, Fig. 74.)

LONDON DEATHS FROM SMALLPOX PER 100,000

1660–1679	417	1850–1860	25
1728–1757	426	1861–1870	16
1771–1780	502	1871–1880	23.4
1801–1810	204	1881–1890	4.5
1831–1835	83	1891–1895	2.0

(The data are in part from Farr's "Vital Statistics.")

In Sweden a law was passed in 1801 permitting doctors to vaccinate against smallpox. This re-

mained in effect until 1816 when everyone was required to be vaccinated who had not previously been successfully vaccinated. The following table shows the highest, the lowest, and the average annual death rate per 100,000 inhabitants in the periods before vaccination was allowed, when it was permitted, and when it was compulsory.

	Highest		Lowest		Average
	Year	Rate	Year	Rate	
Before vaccination 1774–1880	1779	722.7	1786	31.1	200.8
Vaccination permitted 1801–1815	1801	257.0	1814	12.1	63.1
Vaccination compulsory	1875	93.5	1846	0.05	17.3

Vaccination usually gives protection from attacks of the disease for several years, but not for life. One should be vaccinated in very early childhood, again in early school age and then every seven years. These later vaccinations may not take, but it is advisable thus to make sure that one is immune.

Smallpox usually comes in epidemics at varying intervals. One never knows when an epidemic will break out. Montreal, Canada, had an epidemic of smallpox in 1885 and 1886 when 3164

persons died of the disease. The city of Boston, Massachusetts, had an outbreak in 1752. There was then a population of 15,684 and 5998 cases of the disease. Contrast this with present conditions. Massachusetts now has compulsory vaccination. With a population of nearly 4,000,000 people, there have been an average of only 40 cases per year in the last ten years and less than one death per year.

It is in the intervals between epidemics, when the disease is relatively rare that people grow careless, neglect vaccination, and so pave the way for the disease to run violently for a few months or years until people are again scared into being vaccinated. At the close of the Spanish–American War when the United States took over from Spain the government of the Philippines there were in the island annually about 40,000 deaths from smallpox. Vaccination was immediately instituted and in 1916 there were only 239 deaths from the disease. But the natives grew careless as the fatalities from the disease decreased and vaccination was neglected. In 1916–19 came a violent outbreak with 60,447 deaths. Then vaccination was again enforced and by 1923 the prevention had become so effective that only four deaths occurred from smallpox.

It is not claimed that vaccination is an absolute preventative. Some people will catch the disease

even after recent vaccination, but they are very few, and even in them the disease runs a fairly mild course. Vaccination does tremendously reduce the number of cases and the mortality from the disease.

Thus in Detroit, Michigan, in 1920, smallpox broke out, coming in apparently from Windsor, Canada, just across the Detroit River. From April 1st to June 30th there were 482 cases.

380 had never been vaccinated
21 had been vaccinated, but 8 years or more earlier
8 had been vaccinated, but after exposure to infection
71 had been vaccinated, but unsuccessfully
2 caught the disease, although recently vaccinated

482

In this outbreak in Detroit there were in all 1610 cases, with 163 deaths. Of the fatal cases, 142 had never been vaccinated and 19 had been vaccinated ten or more years before they were taken with the disease. Not a single death occurred among those who had been vaccinated within five years. In an epidemic in Los Angeles, California, in 1924, there were 3461 cases, of which 3375 had never been vaccinated; not a single death occurred among the vaccinated.

In Sheffield, England, in 1887–1888, there was an outbreak of smallpox with 4677 cases and 496

deaths. Among vaccinated children under 10 years of age, 1.7% of the cases proved fatal, but 43.9% of the cases among unvaccinated children under 10 years of age resulted in death. Of the vaccinated persons over 10 years of age who were sick of smallpox 5.1% died, while 54.2% of the unvaccinated cases were fatal. These figures and similar ones for smallpox epidemics in other English cities are shown below:

	Cases	Deaths	Per Cent of Cases Fatal Among			
			Vaccinated		Unvaccinated	
			Under 10	Over 10	Under 10	Over 10
Sheffield 1887–88	4677	456	1.7	5.1	43.9	54.2
Dewsbury 1891–92	1012	110	2.2	2.3	32.1	18.7
Leicester 1892–93	357	21	0	1.0	14.0	7.8
Warrington 1892–93	661	62	6	6.4	67.5	33.3
London 1892–93	2353	182	0	2.3	26.7	20.9
Gloucester 1895–96	1979	434	3.8	10.0	41.0	39.7

The following table shows the percentages of vaccinated and unvaccinated people who were living in houses where smallpox broke out and who caught the disease:

	Vaccinated		Unvaccinated	
	Under 10	Over 10	Under 10	Over 10
Sheffield	7.9	28.3	67.6	53.6
Dewsbury	10.2	27.7	50.8	53.4
Leicester	2.5	22.2	35.3	47.6
Warrington	4.4	29.9	54.5	57.6
Gloucester	8.8	32.2	46.3	50.0

Note the much larger percentage of those over 10 years of age than under 10 who caught the disease in spite of having been vaccinated. They had not been revaccinated as they grew older, trusting to the one vaccination of early childhood for protection.

The four million soldiers in the American Army during the World War were all vaccinated. There were only 8 deaths from smallpox, although the troops were often in regions where the disease was prevalent. This is a death rate of 0.2 per 100,000. During our Civil War when the soldiers were not all vaccinated, the death rate was 71.1 per 100,000, more than 350 times as great.

SMALLPOX IN THE UNITED STATES

It must be said to our shame that the United States leads the civilized countries of the world in the number of cases of smallpox. In 1923–24

we had 55,538 cases, exceeded only by India. One-fifth of all the cases of smallpox in the civilized world were in the United States. In 1925 there were in the United States 41,643 cases and in 1926, 33,752. There were eleven states each of which had a larger number of cases than eighteen European countries combined. We had twice as many cases as all of Europe.

The contrast between the number of cases in those states with laws making vaccination compulsory and the ones where such laws do not exist is very marked. Laws compelling vaccination as a prerequisite of school attendance in California were repealed in 1911 and all compulsory vaccination laws were repealed in 1921. In the years 1921–26 California reported 26,985 cases of smallpox and 392 deaths. In the same period, Massachusetts, where vaccination is compulsory, had 64 cases with 2 deaths, and the population of Massachusetts exceeded that of California by 400,000. In the eleven states and the District of Columbia, having compulsory vaccination, there were in the same period 20,973 cases and 189 deaths. These had a population of 36,680,000 in 1926. The remaining states had 283,072 cases and 3521 deaths. Their population was 80,267,000. In other words, the states that do not compel vaccination at all times had 13.5 times as many cases of smallpox and 18.6 times as many deaths from the disease as those states that have adequate

compulsory vaccination laws, although they have only 2.2 times the population of the latter states.

BANISHING THE MOSQUITO AND MALARIA

THE effect of the discoveries of the real cause of malaria and of the part played by mosquitoes in the transmission of the causative agent is nowhere better seen than in Italy. It was in this country, it will be recalled, that some of the most critical experiments were made which proved the mosquito a necessary agent in passing the disease from person to person. Remember that, although in 1880 Lavaran found the protozoan parasite was always present in the blood corpuscles of persons suffering from malaria, Ross did not announce until 1898 his discovery that the mosquito was the host in which this parasite developed. The Italian, Grassi, that same year proved that what Ross had found true for the malaria organism of birds was also true for the one in man. In 1900, those experiments were conducted in the swampy regions about Rome and in England, which showed that one might live in a malarial district without catching the disease if the person were protected from mosquito bites and that the bite of an infected mosquito would induce malaria in regions ordinarily quite free from the disease. About the year 1900, we should have expected to see a marked decline begin, in the death rate from malaria.

There is given herewith a graph (Fig. 78) show-

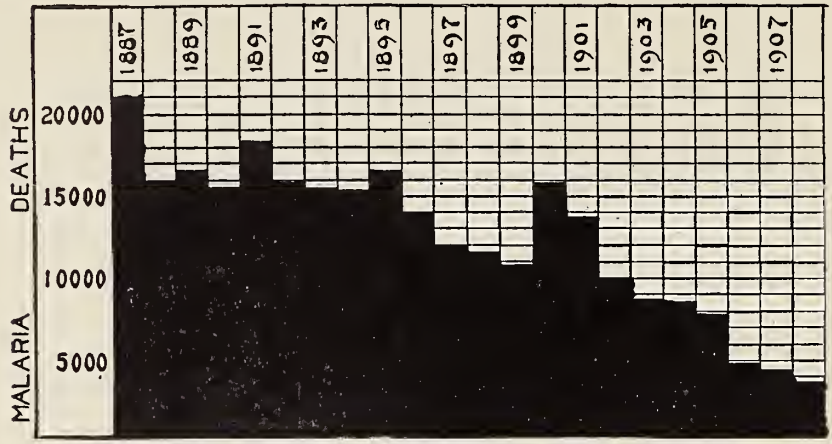


Fig. 78. Mortality from malaria in Italy

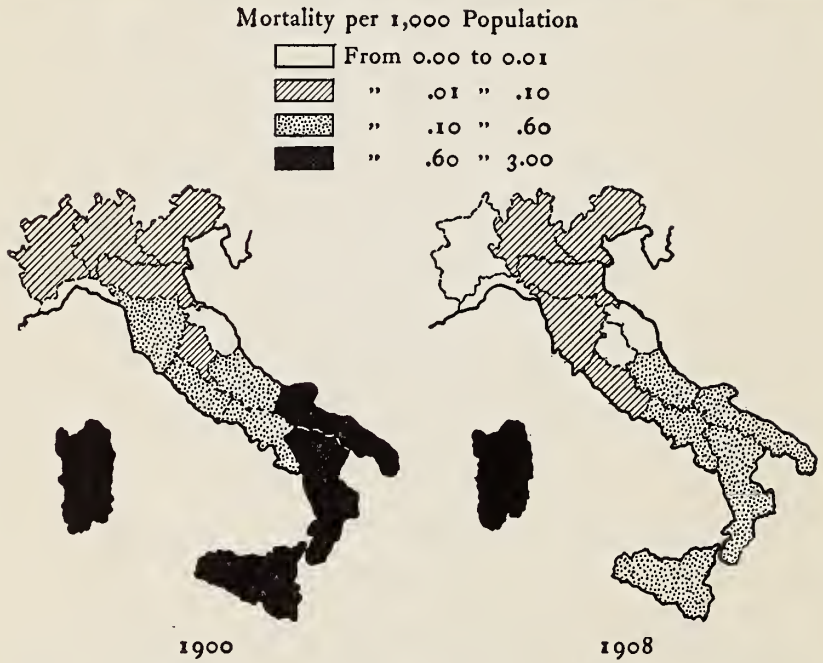


Fig. 79. Mortality from malaria in Italy [From American Museum of Natural History Guide Leaflet 48]

ing the number of deaths from malaria in Italy from 1887–1908, also a map (Fig. 79) showing

how the death rate from this disease is being reduced. Only in the southern portion of Italy where the climate is constantly very mild, thus favoring the activity of the mosquito all the time, is the death rate continuing high.

AVERAGE DEATH RATE IN ITALY PER 100,000 POPULATION FOR 5-YEAR PERIODS

1894–1898	43.9	1904–1908	17.3
1899–1903	36.1	1909–1913	10.0

The real seriousness of malaria is not to be measured by the death rate, however. A great many persons suffer from it, who while they do not die of it, are constantly too sick to work. The disease reduces their earning capacity, their vitality and makes them miserable much of the time. Such persons often die of other diseases which they catch because they are so run down by malaria. Celli, an Italian physician, estimated that in 1900 while there were nearly 15,000 deaths from the disease in Italy, there were about 2,000,000 people sick with the disease. In the State of Mississippi in 1914 there were 116,788 cases, but only 1030 deaths. In 1911, Alabama had 70,000 cases, but only 770 deaths.

In 1914, the death rate in Mississippi was 60.7 per 100,000. Think, then, what such death rates as the following must mean in the number of persons sick with the disease: Dunklin County,

Missouri, one of the worst malarial districts in the United States, 1911–14, 296.7 per 100,000; Philippine Islands, 1909–13, 397.5; Nicaragua, 1908–11, 725.7; Island of Mauritius, 1910–14, 1176.9.

In the United States (registration area) the death rate from malaria was:

1890	19.2	1910	2.6
1900	8.8	1920	3.6

It must be remembered, however, that the registration area at these periods did not include many of the southern states in which malaria is very prevalent. The rise in the rate in the last decade given is largely due to the inclusion of more such states.

The south, however, has naturally profited most by the discoveries that make control of malaria possible as is seen from the death rates in a number of southern cities.

	1900–1904	1910–1914
Richmond, Va.	20.2	1.8
Wilmington, N. C.	222.9	40.3
Charleston, S. C.	62.4	30.9
Savannah, Ga.	132.3	78.6
Jacksonville, Fla.	170.6	34.0
Key West, Fla.	32.5	2.0

Similar contrasts are to be seen in foreign cities in the warmer regions of the earth.

	1900–1904	1910–1914
Havana, Cuba	49.0	2.4
Rio de Janeiro, Brazil	152.9	20.7
Manila, Philippine Islands	98.4	20.6
Calcutta, India	676.5	291.8
Rome, Italy	16.6	8.9
Hong Kong, China (British)	126.5	83.4
Colombo, Ceylon	244.4	37.0

The death rate for each year 1900–14 is shown in the graphs (Fig. 80) for some of these cities. The decline is even more strikingly displayed than it is in the figures above.

Of course, Americans are proud of the results achieved in the Panama Canal Zone during and after the construction of the canal. The death rate from malaria per 10,000 employed persons was, during the period of French construction, 150.9. The United States began work in 1906. The mortality rate among the employees steadily went down as follows:

1906	87.8	1912	3.9
1907	39.1	1913	3.7
1908	16.6	1914	1.6
1909	11.0	1915	2.6
1910	9.8	1916	0.9
1911	9.6		

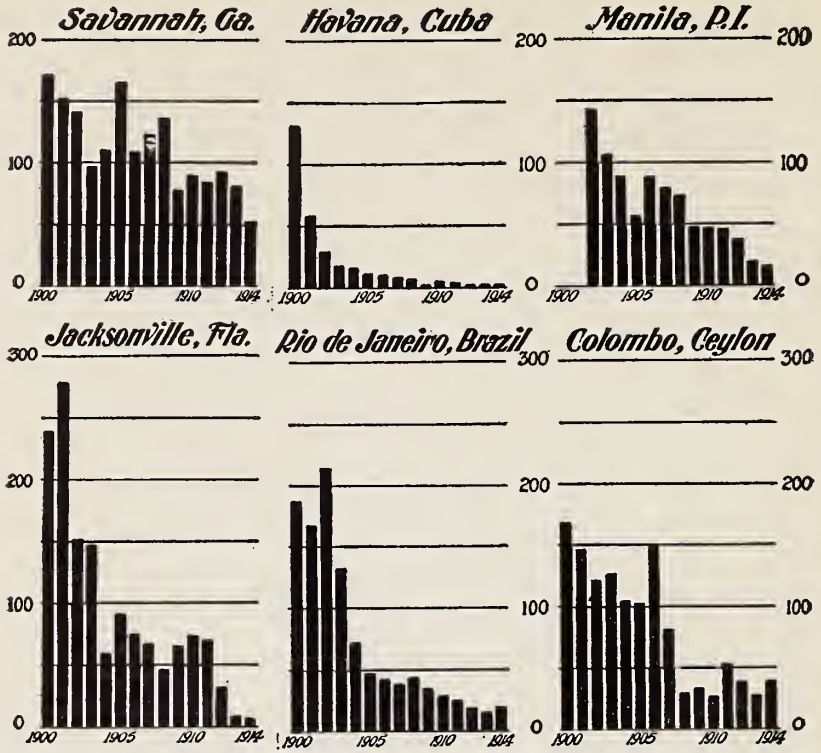


Fig. 80. Decline in Death Rate per 100,000 population (Malaria) [From a Plea and a Plan for the Eradication of Malaria throughout the Western Hemisphere by Frederic L. Hoffman, read before the Southern Medical Association, Atlanta, Ga., 1914.]

DRIVING YELLOW FEVER FROM THE WORLD

THERE is no more thrilling victory in the whole battle against disease than that of the eradication of yellow fever from the world. Remember that in 1900 it was shown experimentally that the germ was carried by one particular kind of mosquito. Patients suffering from the disease were screened so that the mosquito could not attack them, and the breeding places of the mosquitoes, usually water standing about houses, were cleaned up.

The map already given shows the contrast in the prevalence of the disease in the western hemisphere during 1900 and during 1925 (p. 124). In the latter year only three cases of yellow fever were to be found in the whole western hemisphere.

The disease first appeared in the United States at Boston in 1656. It was apparently brought in by sailors from a British squadron of war vessels recently arrived from the Barbados. Since then the disease has assumed epidemic proportions 95 times in various sections of this country. Seventy-six times the infection has come from outside sources, usually brought in by ships; 71 times it has been traced to the Barbados, where the disease was usually very prevalent.

These frequent epidemics were so startling that many of them were described in local publications. A quotation has already been given from Matthew Cary's description of the epidemic in Philadelphia in 1793 when a tenth of the population of that city died of the disease (p. 115). Another epidemic struck the same city in the months of August, September and October, 1798, when 3645 people died. This is described by Thomas Condie in a pamphlet which he and Richard Folwell published in Philadelphia in 1799. It is entitled "A History of the Pestilence, commonly called Yellow Fever, which almost desolated Philadelphia in the months of August, September and October, 1798." William S. Forest was the author of a book of over

three hundred pages entitled "The Great Pestilence in Virginia; being an historical account of the origin, general character and ravages of yellow fever in Norfolk and Portsmouth in 1855." The book was published by J. B. Lippincott & Company in 1856. The disease was brought in by the steamer *Ben Franklin* from which it escaped, according to Forest, by "the dissemination of the concentrated poison discharged from the sweltering hold of the filthy ship." The boat came from St. Thomas where "the fever was raging awfully when she left." There was yellow fever on the ship, in spite of which she was allowed to dock and unload her cargo about the middle of June. The early cases of yellow fever broke out in the immediate neighborhood of this dock and became constantly more numerous. Hoping to check the disease, Barry's Row, a group of ramshackle buildings where many of the cases occurred, was burned down August 9th, but to no avail. Many of the inhabitants left the city. Mr. Forest writes "a stampede has taken place among us. Our city looks deserted. Thousands of people have fled . . . and business is almost entirely suspended. . . . It was too late to fly: the venom had entered the blood and they lay down but to die." One out of every three inhabitants in Portsmouth and Norfolk died of the disease. Four hundred were buried in Norfolk in one week.

James J. Waring described the epidemic at

Savannah, Georgia, in 1876. The pamphlet was published by the Morning News Printing House of that city in 1879. A previous bad epidemic had occurred in 1820 when 729 had died out of a population of 3000 and again in 1854 when 1040 died out of a population of 6000. The population in each case was greater than the figures given, but a large percentage fled immediately when the epidemic broke out. Dr. Earnest Hardenstein of Vicksburg, Mississippi, wrote a pamphlet on the epidemic of 1878 in the Mississippi valley. This was published by J. S. Ross at New Orleans. In the latter city from July to December, there were 4046 deaths and 18,576 cases. In Memphis, Tennessee, out of about 15,000 cases, there were 5130 deaths and in Vicksburg, Mississippi, 6500 cases and 988 deaths. In some 34 cities in the Mississippi valley there were nearly 70,000 cases and over 16,000 deaths.

It is pitiful to see how the doctors of those early epidemics groped around in the dark, trying to discover the cause of the disease. The New York epidemic of 1668 was thought to have been caused by poisonous exhalations given off from a cargo of rotting coffee recently brought in from Brazil in a leaky ship. When the epidemic broke out in Savannah, Georgia, Pasteur had just shown that decay is caused by bacteria. The rice fields and swamps in the neighborhood of the city had recently been subject to flood and the vegetation

was killed. When the flood subsided, this plant material rotted and the stench was very bad. This was thought to be the probable cause of the disease. After the bad epidemic in the Mississippi valley in 1878, Congress appointed a commission of experts to study the outbreak. They reported that yellow fever is caused by a specific poison of unknown origin and nature. They thought it was probably organic and was endowed with the power of growth and reproduction. Since the disease was confined to certain localities, they thought there must be local conditions favorable to the development of the poison but what these conditions were they confessed they did not know. They added, however, "the discovery of these local conditions will be a great boon to humanity," a prophecy that was fulfilled when the mosquito was found to be the active agent in spreading the disease and the local conditions necessary were discovered to be places where this mosquito could breed.

YELLOW FEVER IN OTHER PARTS OF THE WORLD

THE facts given make it clear what a dreadful pestilence yellow fever was in the United States. It was equally dreaded all through tropical and subtropical countries. Now notice the sudden cessation of yellow fever when those sick with the disease were kept screened and when the breeding places of the mosquito were destroyed. For-

tunately this yellow fever mosquito does not breed extensively in swamps, as does the malarial mosquito, but in water vessels or little pools about houses. It is fairly easy, therefore, either to screen or to get rid of such places. Such measures were adopted in Havana in 1900, in Rio de Janeiro, Brazil, in 1906, in Guayaquil, Ecuador, in 1921.

Havana is a city of about 300,000 population. There follows a table showing the deaths from yellow fever, in Havana, for a period of years:

Year	Deaths	Year	Deaths	Year	Deaths
1891	356	1897	858	1903	0
1892	357	1898	136	1904	0
1893	496	1899	103	1905	2
1894	382	1900	310	1906	12
1895	553	1901	18	1907	5
1896	1282	1902	0	1908	1

Since the last date no cases have occurred except an occasional one brought in by a ship.

Here are the figures for Rio de Janeiro with a population of about 1,000,000:

Year	Deaths	Year	Deaths	Year	Deaths
1891	4456	1902	94	1906	49
1896	2929	1903	584	1907	39
1900	344	1904	48	1908	4
1901	299	1905	289		

Again since 1908 there have been no cases except an occasional one brought in by a ship. There is given a graph (Fig. 81) showing the drop in the number of deaths in Cuba. It is suggested that similar graphs be constructed from the figures given for Havana and Rio de Janeiro.

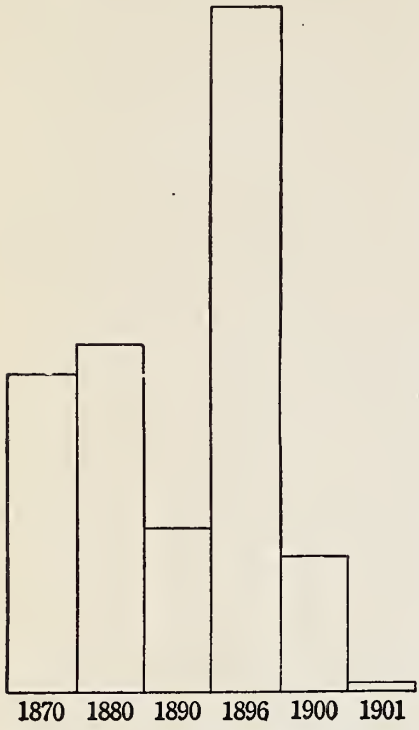


Fig. 81. Graph of Yellow Fever in Cuba

TUBERCULOSIS

For many years tuberculosis caused more deaths in the United States and in the world as a whole than any other one disease. Over 1,000,000 people die annually from it, the world over. This means there are about two deaths a minute. The death rate from tuberculosis has been steadily falling, however, while that from some other diseases has been rising, so that in the United States during the 10-year period, 1914–23, tuberculosis is third on the list of the ten most deadly diseases. These ten causes of death and the average death rate from them for the 10-year period is given below:

AVERAGE DEATH RATE PER 100,000 POPULATION FOR 1914–23

All Causes, 1352.76

Heart disease	164.0
Influenza and pneumonia	151.9
Tuberculosis (all forms)	126.24
Nephritis	102.0
Congenital malformation and diseases of early infancy	87.1
Cerebral hemorrhage, softening, embolism and thrombosis	86.21
Cancer and other malignant tumors	83.15
Accidents	76.84
Bronchitis and bronchial pneumonia	64.08
Diarrhœa and enteritis	62.38

These ten total 1003.90 per 100,000 and account, therefore, for nearly three fourths of all the deaths. There is given also a graph (Fig. 82) showing the death rate from these ten causes. Turn also to the appendix on page 313, where there is given a graph showing the deaths per 100,000 of the population in the United States for each year from 1915 to 1924. The strikingly

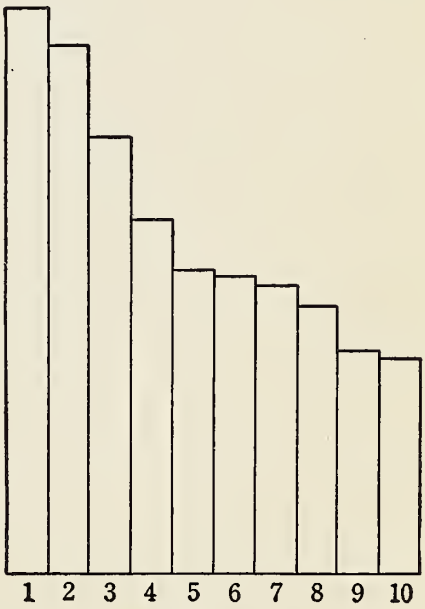


Fig. 82. Graph of death rate from ten most important causes, average of ten-year period in United States. Order as in table above

TUBERBULOSIS DEATH RATE PER 100,000

Year	Mass.	Eng. and Wales	Year	Mass.	Eng. and Wales	
1875	416.1	299.4	1900	251.9	190.1	
1876	380.4	286.5	1901	234.5	180.5	
1877	374.7	284.2	1902	221.0	173.7	
1878	365.6	292.3	1903	209.1	173.9	
1879	358.6	276.1	1904	219.8	178.1	
1880	378.9	270.8	1905	214.7	163.0	
1881	396.4	253.0	1906	212.9	164.5 *	
1882	387.7	258.1	1907	178.6	160.6	
1883	385.6	259.1	1908	166.2	158.5	
1884	378.0	257.8	1909	160.7	152.4	
1885	377.1	243.0	1910	162.7	142.2	
1886	366.8	247.3	1911	154.4 crude	145.5	
1887	361.8	228.3	1912	148.1 "	135.1	
1888	350.3	222.8	1913	145.0 "	131.3	
1889	331.9	225.8	1914	143.1 "	131.9	
1890	327.5	237.6	1915	141.6 "	150.9	Civilian population only
1891	312.5	230.0	1916	149.2 "	161.0	
1892	316.0	214.2	1917	145.9 "	182.8	
1893	306.6	214.2	1918	159.0 "	195.0	
1894	295.8	197.2	1919	128.0 "	122.7	
1895	291.8	205.9	1920	113.8 "	108.9	
1896	286.4	189.2	1921	98.5 "	113.1	
1897	271.8	192.4	1922	93.8 "	112.7	
1898	261.3	190.8	1923	88.5 "	106.2	
1899	253.0	190.3	1924	.....	105.8	

* To this point figures taken from "Tuberculosis in the United States," Census Bureau, 1907.

high death rate from influenza and pneumonia in 1918, due to the epidemic of that year, was found not only in the United States, but the world over.

Since statistics are to be had for the registration area of the United States only since 1900, data must be obtained from other sources for the earlier periods. There is given a table covering a fifty-year period showing the death rate from all forms of tuberculosis in England and Wales and in the State of Massachusetts.

These figures are also put in graph form in the chart on page 198 (Fig. 83). A similar graph is given for the city of Chicago (Fig. 84). You will see from a study of these that all through the years given there has been a steady decline in the deaths from tuberculosis, except for the period of the war.

For a very long time tuberculosis had been recognized as a much dreaded human disease. Hippocrates (B.C. 460–377), who has before been referred to as the "father of scientific medicine," described tuberculosis and recognized it as the disease that proves fatal to the largest number of people. Isocrates, who lived about the same time, recognized the disease as a contagious one. Apparently, however, the contagious nature of the disease was ignored until the early nineteenth century. It remained for a French physician, Villemin, to demonstrate by experiments on animals the contagious nature of the disease. You

will remember that it was in 1882 that Robert Koch announced the discovery of the tubercle bacillus and gave his proof that it was the cause of the disease.

This bacillus attacks many of the organs of the

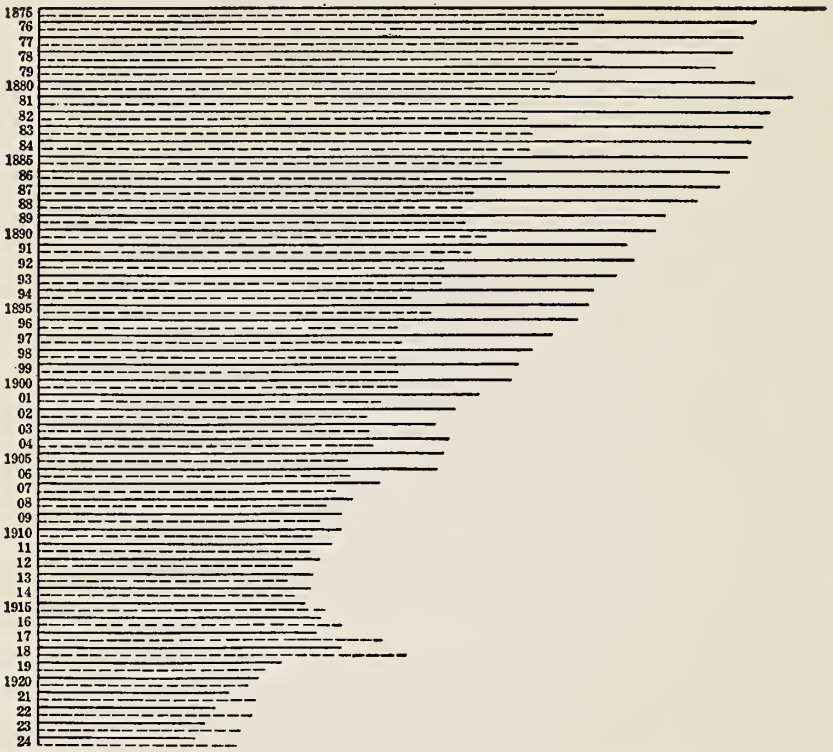


Fig. 83. Death rate per 100,000 population from all forms of tuberculosis in Massachusetts (solid lines) and England and Wales (dotted lines)

body. The most prevalent form of tuberculosis is that of the lungs, commonly called consumption, but the germ attacks the covering of the brain (*meninges*), the joints, the vertebrae, the intestine, the lymph glands, and, sometimes, it is very gen-

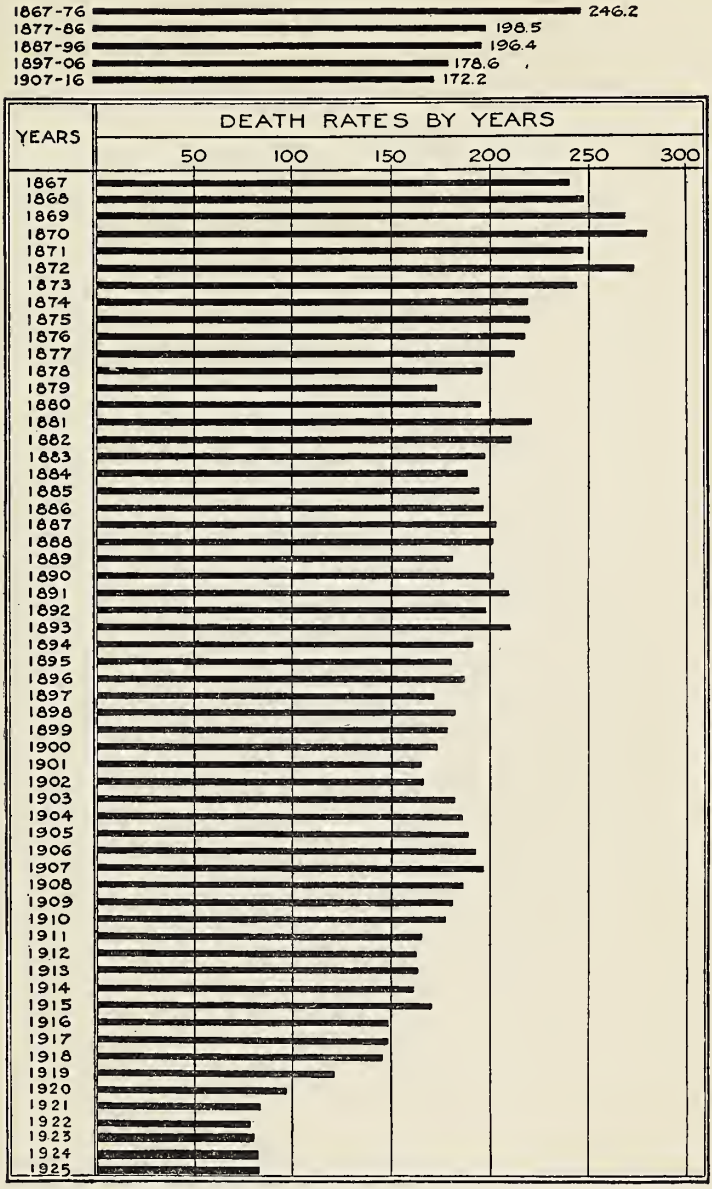


Fig. 84. Graph showing the decline in the death rate from tuberculosis in Chicago. (Death rates per 100,000 of the population)

erally spread through all the organs of the body. There is given below a table showing the death rate in England and Wales from 1847 to 1924 from tuberculosis of the respiratory organs:

Note the steady decline in the death rate. By 1924 it was reduced to almost one-fourth the rate of 1847. We see in this tabulation and in the preceding graphs no marked fall in the death rate immediately following Koch's discovery of the germ of tuberculosis. We have shown that after the discovery of the diphtheria germ and of the antitoxin there was a marked decline in the death rate from diphtheria. Similarly following the discovery of the germ of yellow fever and of the fact that it is carried exclusively by the mosquito, there was a corresponding decline in the prevalence of yellow fever. The decline in the death rate from tuberculosis has, however, been only a little more rapid since Koch's discovery than it was before.

Evidently this contrast in the effect of the discovery of the germ of tuberculosis and of the other discoveries whose effects we have previously discussed requires comment. Tuberculosis is a very widespread disease. Probably on an average one out of every fifty persons has active tuberculosis. Since such persons are frequently giving off the bacilli of tuberculosis from their lungs during respiration or from other organs in their excretions these germs are constantly present where people are gathered in crowds, even in small

ANNUAL REPORT OF THE CHIEF MEDICAL OFFICER OF THE MINISTRY OF HEALTH FOR THE YEAR 1924

London, 1925. His Majesty's Stationary Office. Death rate per 100,000 for tuberculosis of the respiratory system. 1847–1924. England and Wales.

Year	England and Wales	Year	England and Wales	Year	England and Wales
1847	318.9	1875	231.3	1902	122.9
1848	304.6	1876	222.7	1903	119.6
1849	294.0	1877	218.4	1904	122.8
1850	270.2	1878	221.6	1905	113.0
1851	281.7	1879	212.4	1906	113.8
1852	285.8	1880	196.2	1907	112.5
1853	307.4	1881	192.3	1908	109.9
1854	283.3	1882	194.5	1910	98.8
1855	286.4	1883	197.4	1911	103.5
1856	264.9	1884	191.7	1912	99.3
1857	269.0	1885	185.5	1913	96.1
1858	268.3	1886	181.7	1914	99.2
1859	264.3	1887	168.5	1915	115.5 *
1860	265.2	1888	163.4	1916	123.0 *
1861	267.5	1889	163.7	1917	138.4 *
1862	259.6	1890	174.8	1918	152.2 *
1863	256.9	1891	165.9	1919	95.3
1864	263.6	1892	151.7	1920	84.3
1865	265.1	1893	150.9	1921	84.6
1866	271.7	1894	141.9	1922	84.9
1867	265.3	1895	142.6	1923	79.8
1868	244.1	1896	132.7	1924	80.1
1869	246.1	1897	135.6		
1871	245.7	1898	132.5		
1872	238.4	1899	133.9		
1873	230.2	1900	133.7		
1874	218.5	1901	126.3		

* Civilian population only.

crowds. Very few persons escape infection from tuberculosis at some time in their lives, most commonly in childhood, but often at other times as well. Thanks to the efficiency of our white blood corpuscles and to other means of defense in the body, the disease is checked before it gets a good start. Tubercular people are not confined in hospitals as are those suffering from some contagious disease like smallpox, but they are constantly about us as sources of the disease. Furthermore, there has not yet been discovered a specific cure for tuberculosis. In the case of smallpox, vaccination is a preventative, as is the toxin-antitoxin treatment in the case of diphtheria. Similarly we can prevent typhoid with the injection of a specific serum. Robert Koch thought he had discovered a cure for tuberculosis in tuberculin, but he was mistaken. Since his time a dozen or more cures for the disease have been announced, but none of them has been found effective. The germ of tuberculosis does give off a poison as it grows in the tissues or in the cultural media, but it is not this poison that kills the patient. The germ is a true parasite, eating up the tissue on which it lives and it is this destruction of a vital organ that causes death. The decline in the death rate from tuberculosis, therefore, is due first to the means at our command for discovering the disease in its early stages and so initiating curative treatment; second, to the hos-

pitalization of patients in the more advanced stages of the disease so that they will not be sources of infection to the entire community; third, to greater care in the observation of rules of hygienic living among the general population.

With all these discoveries of the causes of disease and the means of preventing them, one wonders that there are still so many people sick. The English Minister of Health said (1924) that the insured workers of England lose over 20,000,000 weeks of work every year through sickness and the insured workers are only a part of the total number. This is equivalent to 375,000 workers being idle the whole year. This means a direct loss in wages for England and Wales of over $700,000,000. The annual loss in the United States is figured at over $3,000,000,000. This does not take into account the money spent for doctors and medicine. It gives an idea, only as you use your imagination, of the suffering and misery produced.

There are a good many diseases whose causes and means of prevention are not yet known. The young men and women of today have still plenty of opportunity to make discoveries like those about which we have been learning. But still much of this sickness, misery, and loss of wages is preventable. People, both individually and collectively, are careless. They will not take good care of themselves and observe the precautions

they know they should take. Society often does not enforce the health regulations that are legally required. People are forced to live in crowded, unhealthy quarters because we are so slow to act upon our knowledge that such places are disease centers from which death moves on to the well-to-do sections, often with more dreadful results than in the slums themselves. Part of this loss through sickness is due to ignorance. Many people still do not know the facts that have been presented in this book thus far. It is to get such information to those who read and to inspire them to tell it to all their acquaintances that such books are written. For all the work of these discoverers will have been in vain unless the knowledge gets to every one, so that they will know how to prevent and to cure disease, and will act upon this knowledge.

CHAPTER VII

EARLY IDEAS OF THE HUMAN BODY AND ITS WORK

OUR DEBT TO THE PAST

MANY PAGES have been used in tracing the growth of one idea—that diseases are largely due to the presence of specific tiny living creatures in our bodies that may destroy our essential organs, or that may develop poisons within us that kill us. We have shown that in many cases science has discovered how to combat these germs of disease and so cure, or, better still, prevent disease. This discovery of the germ nature of disease and of the consequent methods of cure or of prevention has been one of the most important contributions that science has made to man's health. The applications of this principle are familiar on every hand. We wash our hands before meals, clean our teeth often, avoid crowded, badly ventilated rooms to lessen the danger from bacteria, often, for the same purpose, we send persons with dangerous diseases to hospitals.

(Write out a list of other things that are done to prevent the spread of disease germs.)

But this discovery of the germ nature of disease is by no means the only contribution science has made to health. Science, through a long series of discoveries, has enabled man to understand how his body works, how it moves, feels, digests, breathes, and so it has taught him how to take proper care of himself. The elementary textbooks on physiology and hygiene studied in our schools today would have been stunning revelations to even the greatest scientists of two or three hundred years ago. These facts regarding the structure of the body and the way its various organs, work have been found out slowly, one at a time by many patient investigators. The story of such discoveries runs back through hundreds, even thousands of years:

We are, all of us . . . Children of our Fathers. What we are is in part only of our own making, the greater part of ourselves has come down to us from the past. What we know and what we think is not a new fountain, gushing fresh from the barren rock of the unknown at the stroke of the rod of our own intellect; it is a stream which flows by us and through us, fed by the far-off rivulets of long ago. As what we think and say today will mingle with and shape the thoughts of men in the years to come, so in the opinions and views which we are proud to hold today, we may, by looking back, trace the influence of the thoughts of those who have gone before.[1]

[1] "Lectures on the History of Physiology," Sir M. Foster; Cambridge Press, 1901; Lecture I, p. 1.

PRIESTS THE EARLY PHYSICIANS

PRIMITIVE peoples looked upon what we call natural phenomena—the happenings in nature—as the work of the gods. The sun was carried about the heavens from sunrise to sunset in the chariot of a god. The wind was the breath of a god. An awful storm or an earthquake was the work of an angry god or perhaps a demon. Sickness that caused such intense suffering was thought to be due to some evil spirit that took possession of the sufferer. A cure might be effected by scaring away this evil spirit and so the friends of the sufferer danced about him, wearing hideous masks and making terrifying noises (Fig. 85). Better still, one might keep such evil spirits away by wearing some amulet, by keeping horrible images set up about the camp, or by making offerings or sacrifices to these powerful demons. Usually among

Fig. 85. Mask and wooden gloves worn by certain savage tribes [Courtesy The Field Museum of Natural History]

such primitive folk there were medicine men who were credited with skill in the "magic" needed to drive away the evil spirits or to appease the offended demons. These medicine men were held in religious reverence, for they were thought to have power with the unseen spirits of the universe and stood between the people and the gods and demons (Fig. 86).

Fig. 86. Indian Medicine Man [Courtesy the Field Museum of Natural History]

It is not surprising, therefore, to find that in the dawn of civilization the priests served as physicians. This was true in Babylon, Egypt, Israel, and Greece. Apollo was the god of health among the ancient Greeks. When angry he visited plagues and epidemics on mankind; his favor insured health. His son, Æsculapius (Fig. 87), was a skillful healer, so skillful that he was killed by the God Zeus, so legend says, for fear men would cease to die. Later he was worshipped and temples were erected to him beside health-giving mineral springs, on wooded mountains where the invigorating air restored the sick, or in other health-

ful locations. These temples became sanitaria, and the priests of Æsculapius were skilled in treating disease. Among the earliest of these temple attendants were Hygieia and Panacea, daughters of Æsculapius. The sanitaria were known as Asclepieia. A famous one was located at Cos.

Fig. 87. ÆSCULAPIUS [Courtesy N. Y. Academy of Medicine]

In early times the sick had to be taken to the temples to be treated by the priests. This was true not only among the Greeks, but also among the Egyptians, the Jews, and the Babylonians. Later, however, the priests began to go out to visit and heal the sick. Gradually the traveling priest-doctor lost all connection with the temple and became a physician, pure and simple. In Egypt the separate profession of the physician was recognized at least 2500 years B.C.

SKILL OF THE EARLY PHYSICIANS

One marvels at the surgical and medical skill of the priest-doctors even back in the very early times. Yet, of course, man had for thousands of years been learning by the trial and error method what to do in case of illness. The sick man would eat this or that, trying, as animals do, to find something which would give relief; often he failed, but occasionally he succeeded and told his fellows what had helped him.

Primitive man must have learned something of bodily structure as he cut up animals for food, observed their skeletons occasionally seen in woods or fields, or saw the play of muscles and joints in action on his naked fellows. Thus medical lore gradually accumulated. The early priest-doctors knew enough of body structure to set broken bones, to reduce dislocations, to extract painful teeth and to perform some quite complicated operations (Fig. 88). They even gave sleeping potions of drugs like opium, Indian hemp, or hemlock, to prevent the patient's feeling the pain in the operation. In ancient Egypt there were specialists who confined their practice to one sort of disease or to one sort of operation, like the removing of stones from the bladder or the taking of a cataract off the eye. These early priest-doctors knew how to treat and bandage wounds; they prescribed medicines, many of which were obtained from herbs.

Some knowledge of the healing value of plant remedies is found even among savages. Our North American Indians knew the value of golden seal and prickly ash berries for the cure of fevers,

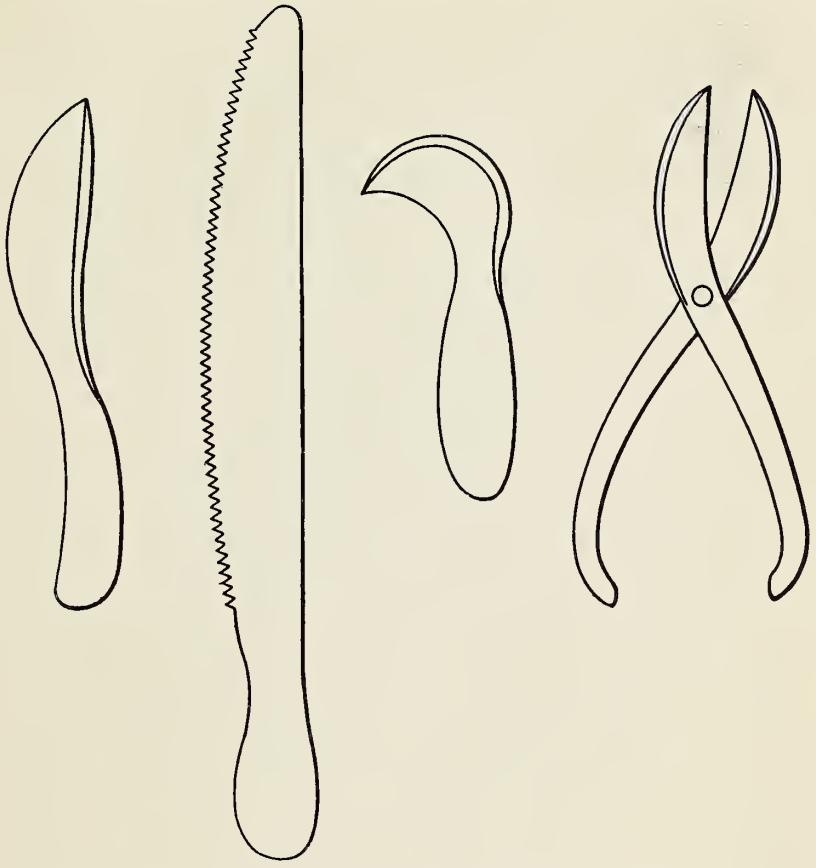


Fig. 88. Tools of the ancient Egyptians
A knife, a saw, a curved knife, shears. Handwrought in bronze. With some such the early surgery was done.

of wild ginger and ginseng for digestive troubles, of lobelia for coughs and colds, of sassafras leaves for healing wounds and the value of many other simples gathered in field and forest.

HIPPOCRATES

Much of this early medical lore has come down to us in the writings of a noted Greek physician,

Fig. 89. Hippocrates [Courtesy N. Y. Academy of Medicine]

Hippocrates, who is often called the "father of medicine" (Fig. 89). He apparently learned

what was known of medicine in his day, and he added to that knowledge much that he himself discovered. He was born on the Island of Cos in 460 B.C. He attended the medical school of Cos, one of the most famous in Greece. He taught and practiced there, but also traveled and studied a great deal. He lived to be about eighty years old, a ripe old age for those days. He evidently knew enough to keep himself in good health for a long time.

He believed that diseases were due to natural causes, not to evil spirits. He was careful to observe the symptoms of disease and so to distinguish between various kinds of diseases. He recorded the patient's rate of respiration, roughly took his temperature and noted his general nutrition; he examined the excretions. In a word, he observed all those indications of disease that the modern doctor can note without any of the modern instruments at his command. He was a skillful surgeon; he washed out wounds with boiled water, covered them with dry dressings and insisted on a clean, light operating room with capable assistants. He wrote books: "On Epidemic Disease," "On Diet in Acute Disease," "On Wounds of the Head," "On Fractures," "On Ulcers," etc. The titles show with what a wide range of subjects he was familiar.

The oath that physicians of his day were compelled to take is known as the oath of Hippocrates,

although it was probably in use long before he lived. Somewhat abbreviated it reads as follows:

I swear that according to my ability and judgment I will keep this oath and stipulation; to reckon him who taught me this art equally dear to me as my parents, to share my substance with him and relieve his necessities if required; to regard his offspring as on the same footing with my own brothers, and to teach them this art if they should wish to learn it, without fee or stipulation, and that by precept, lecture, and every other mode of instruction, I will impart a knowledge of the art to my own sons, and to those of my teachers, and to disciples bound by a stipulation and oath, according to the law of medicine but to none others.

I will follow that method of treatment which according to my ability and judgment I consider for the benefit of my patients and abstain from whatever is deleterious and mischievous. I will give no deadly medicine to anyone if asked, nor suggest any such counsel.

With purity and holiness I will pass my life and practice my art. I will not cut a person who is suffering with a stone, but will leave this to be done by practitioners of this work. Into whatever houses I enter I will go into them for the benefit of the sick and will abstain from every voluntary act of mischief and corruption.

Whatever, in connection with my professional practice or not in connection with it, I may see or hear in the lives of men which ought not to be spoken abroad, I will not divulge, as reckoning that all such should be kept secret.

While I continue to keep this oath unviolated, may it be granted to me to enjoy life and the practice of the art, respected by all men at all times; but should I trespass and violate this oath, may the reverse be my lot.

The oath shows that these early priest-doctors

took their profession very seriously and had high moral standards.

ARISTOTLE

UNDOUBTEDLY the greatest scientist of the Greek world, probably the greatest of the ancient world, was Aristotle. (See Fig. 5.) He has been called the "father of natural history." He was born at Stagira in Macedonia in B.C. 384. He was the son of a physician and was a pupil of Plato, one of the greatest of the Greek teachers.

Aristotle dissected many animals. He performed experiments on them to find out how their parts worked. He studied their development from the egg. He collected and studied plants. Thus he made no mean beginnings in botany, embryology, physiology and comparative anatomy. Through his influence a great center of scientific learning was founded. His method of increasing knowledge by careful observation and experiment set standards that were not improved for many centuries. He also wrote many books on a great variety of scientific subjects. He became the tutor of the young Alexander, who was later to be known as Alexander the Great.

When the crown prince became king he placed at the command of Aristotle thousands of men who were scattered all through Greece and Asia to make observations for him and to collect specimens. When Alexander conquered Egypt he

founded the city of Alexandria and developed there a center of learning, including a great botanical garden, an immense zoölogical collection, a museum, a building for anatomy where human dissections were made, an astronomical observatory, and a library containing thousands of volumes. Thus the influence of Aristotle was passed on as well as that of Hippocrates, for the medical school used Hippocrates' books and methods as the basis of its instruction. It is evident that the facilities of the scientists of that ancient time were quite comparable to those of modern times. Aristotle's works on animals and plants, on physics, and other scientific subjects, included most of the knowledge of his day, much of which was the result of his own original observations and experiments.

GALEN

Still a third man deserves notice, another great physician, Galen (A.D. 131–201) (Fig. 90). He was, for many years, a physician to the Roman emperors; more than that he was a student and an investigator. He was born at Pergamos and studied medicine at Smyrna. It was rarely possible in his day for the medical student in Rome to dissect human bodies, but he did make dissections of animals, particularly pigs and apes. He described the skeletal system and the muscles and made careful studies of the brain. He wrote a

great many medical treatises, some of which merely embodied the current knowledge of his day, but many of which recorded the results of his own studies in anatomy and of his experiments on the functions of the various parts of the body.

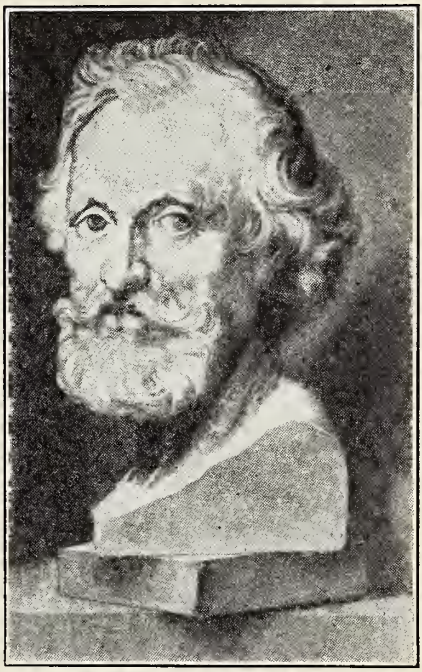


Fig. 90. GALEN. [Courtesy N.Y. Academy of Medicine]

Today some of Galen's ideas about the way the various parts of the body act seem ridiculous yet he had grasped some fundamental ideas in physiology. One is amused at his ignorance, yet amazed that he knew so much. He knew that food is digested in the stomach, yet thought that this was done largely by the heat from the nearby heart. The digested food then went to the liver and was changed to blood. He thus recognized the liver as an important organ, as indeed still earlier scientists had done, some of them even believing it to be the seat of the soul. In the liver, Galen thought the blood was purified, the "black bile," an earthy impurity being taken out, while the yellow bile was stored in the gall bladder. The

liver also furnished the blood with "natural spirits," a principle or property that took care of nutrition, growth, and reproduction.

The blood then went to the heart. Galen knew from his experiments that the heart pumped the blood but he thought the heart sucked the blood in, rather than forced it out. He believed, too, that the blood washed back and forth in the blood vessels, an ebb and flow movement. The blood in the veins carried "natural spirits"; that in the arteries carried "vital spirits." Some of the blood reached the lungs, some of it went to other parts of the body. The heart also drew air into itself from the lungs, and took out of it a principle, "pneuma" he called it, which helped maintain the heart's heat. He had an idea that the heart was a sort of furnace. The heart, too, furnished the blood with "vital spirits," that accomplished most of the activities of the body, peculiar to living things. When the blood reached the brain, the vital spirits were changed to "animal spirits" that went out through the hollow nerves to produce movements and the mental functions. It is interesting to note how Galen sensed that there were important activities going on in the digestive organs, heart, and brain. He did not know exactly what was taking place so he used the term "spirit" to indicate the property developed. The liver added a property of nutrition or "natural spirits," the heart produced a property

that made activity possible, the "vital spirits," and the brain made something that controlled activity, the "animal spirits."

Aristotle thought the brain was a sort of refrigerator that cooled the blood and so controlled the emotions and passions that resided in the heart. We still speak of a person as "hot-headed" if he lacks such control. Galen, however, knew the brain as an organ of nervous control, for he had experimented on the brain, the spinal cord, and the nerves of animals. He even knew there were some nerves that carried sensations, some that caused motion. Still he thought nerves were like strings which when pulled caused muscles to act.

THE BEGINNINGS OF MODERN NOTIONS

For nearly thirteen hundred years after the time of Galen there were almost no additions made to the knowledge of the structure and the action of the human body. This was largely due to the fact that the civilized world was involved in many wars; Greek and Roman culture was largely wiped out by the invasion of barbaric peoples from the north of Europe. Then, too, the Church came to be a mighty power. She assumed the right to govern with firm authority, not only in matters spiritual, but frequently in matters temporal. On the whole, probably such authority was beneficial, but, unfortunately, the same reverence that was demanded for the religious writings sponsored

by the Church, was also claimed for the writings of Hippocrates, Aristotle, and Galen. These came to be regarded by the scientists as verbally inspired and, therefore, the only authoritative sources of scientific knowledge. For hundreds of years most scientists read Aristotle's works and medical students read Galen's works and simply accepted their statements as true for all time. There were, of course, here and there, men who were making observations for themselves and doing some thinking for themselves, but, on the whole, little progress was made during the so-called Dark Ages.

We find, therefore, at the time of the revival of learning in Europe that the ideas of Aristotle, Galen, and others of the old scientists regarding the structure and functions of the human body were still confidently accepted. Moreover, these ideas were not so clear as they had been in the times of the Greeks, for while they were coming down through centuries of ignorance and superstition many wrong notions had become attached to them.

Andreas Vesalius (Fig. 91) was the first to stand in open, successful revolt against the teachings of these ancient worthies and to insist on his right to describe things as he saw them, not as Galen said they were. While Galen made many valuable contributions to knowledge, he could not dissect human bodies and his anatomy of man was full of mistakes. (Anatomy is the science

that deals with the coarse structures of the body.) Vesalius was born in Brussels (Belgium) at the very end of 1514, just as the old year was going out. He was sent to school at Louvain. Even as a lad he dissected animals and early knew he wanted to be a physician. So he was sent to Paris to study. But the Church felt that human beings, even when dead, were too sacred to dissect. Vesalius, however, secured human bones from the graveyards and once pulled down from the gallows on a public road the skeleton of a criminal whose bones the birds had most thoroughly cleaned. This he took by night to his lodgings so that he might study it.

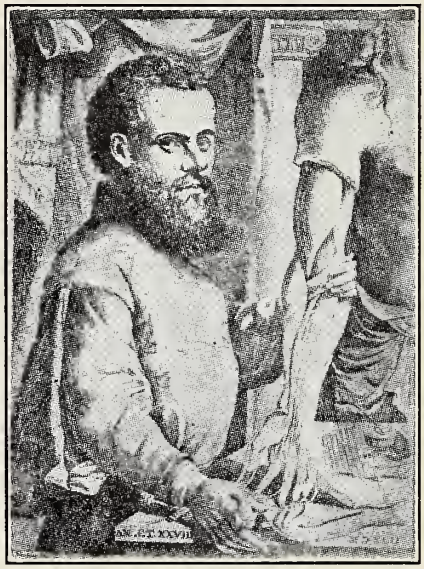


Fig. 91. ANDREAS VESALIUS (Locy)

He learned, however, that at the University of Padua in Venice, then an influential kingdom, dissections of human bodies could be seen. So thither he went as a student in 1537 and that same year he was made a doctor. He became so skillful in dissecting and in demonstrating human anatomy that he was shortly appointed to the chair of surgery and anatomy. For five years he

taught and demonstrated to his students. Meanwhile, he occupied all his spare time in studying the dissections of human subjects that he secured from doctors, from the jails when criminals were executed and even from the graveyards. He studied enthusiastically.

Then he wrote a treatise on human anàtomy and illustrated it with many plates of fine drawings. Do you suppose it was welcomed as a great addition to human knowledge? Not at all. The old fogies still wanted to read their anatomy out of Galen, and did not want a young upstart to tell them it was largely wrong. Many of Vesalius' friends deserted him, even his old teachers. He was called sacrilegious for cutting up human subjects. Fortunately, he could not be cast into prison for such acts, as would promptly have happened in most countries of Europe, because Venice was then an independent and powerful country. In disgust he went off to Spain and became court physician to King Charles V. But his book remained. Some of his students, Fallopius especially, carried on the good work he had begun. Gradually the authority of Galen waned and medical students learned their anatomy from human bodies, not from a book. This more accurate knowledge of the structures of the body paved the way for a better understanding of the functions or uses of its parts.

WILLIAM HARVEY

THE next startling discovery was made by William Harvey (Fig. 92), an Englishman. He was born at Folkstone on the southern coast of England, in 1578. He took his Arts degree at Cambridge and then, in 1593, went to Padua to study medicine. Fabricius, a pupil of Fallopius, was now occupying Vesalius' old chair of surgery and anatomy. Fabricius had discovered the valves in the veins (Fig. 93). On obtaining his degree in medicine, Harvey returned to England and settled to practice in London. In 1609 he was appointed physician to St. Bartholomew's Hospital and later became physician to King Charles I. He was also lecturer in anatomy at the Royal College of Physicians and Surgeons. The remarkable thing he did was to demonstrate the circulation of the blood. He studied the hearts of many animals, watching the pulsating movements and the movements of the blood in the attached vessels. One animal in particular was instructive, a shrimp that lives in the shallow waters in the sea.

Fig. 92. WILLIAM HARVEY [Masters]

He knew this animal from boyhood days. It is so transparent one can see the heart beat and the blood movements without having to cut the animal open.

First, Harvey realized that the strong muscular ventricles of the heart of the higher animals force the blood out into the arteries. (See Fig. 94, The Heart, and Fig. 95, The Course of the

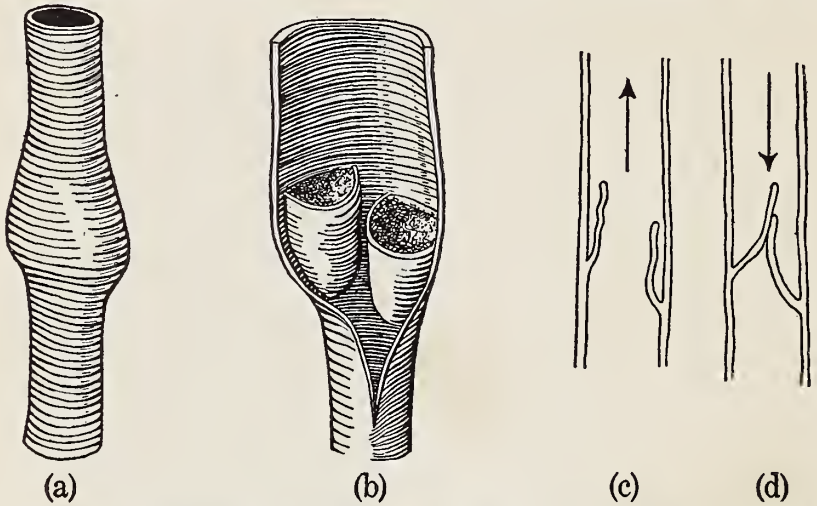


Fig. 93. (*a*) Exterior of part of vein with valves in operation. (*b*) Such a vein opened to show valve flaps distended. (*c*) Valve flaps when blood is flowing toward the heart. (*d*) Valve flaps when blood backs up (same as *a* and *b*)

Circulation.) The heart is a force pump, not a suction pump. Then he determined by observation about how much blood the heart sends into the arteries at each beat. When he multiplied together this quantity, the number of times the heart beats per minute, the number of minutes per hour, and the number of hours per day, he saw that the total was so great that the food could not produce blood fast enough to send so much

of it through the body. The blood, therefore, must return to the heart constantly to be sent out again and again. So he concluded that the blood circulates. He saw that the valves in the veins which his teacher, Fabricius, had discovered

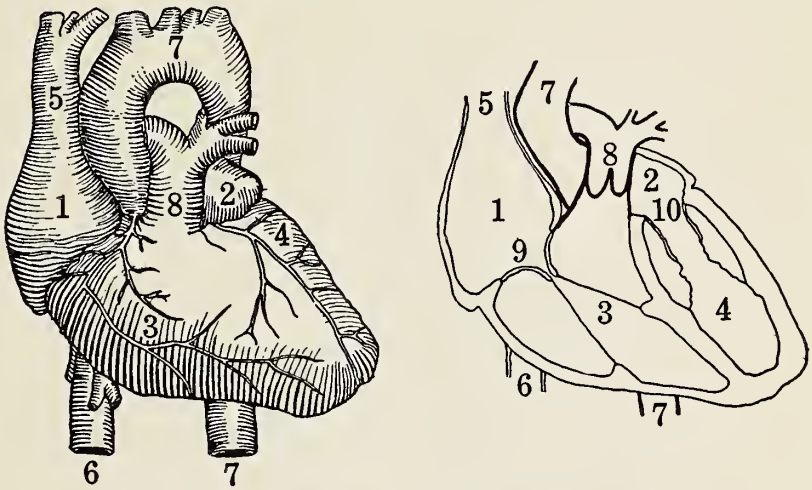
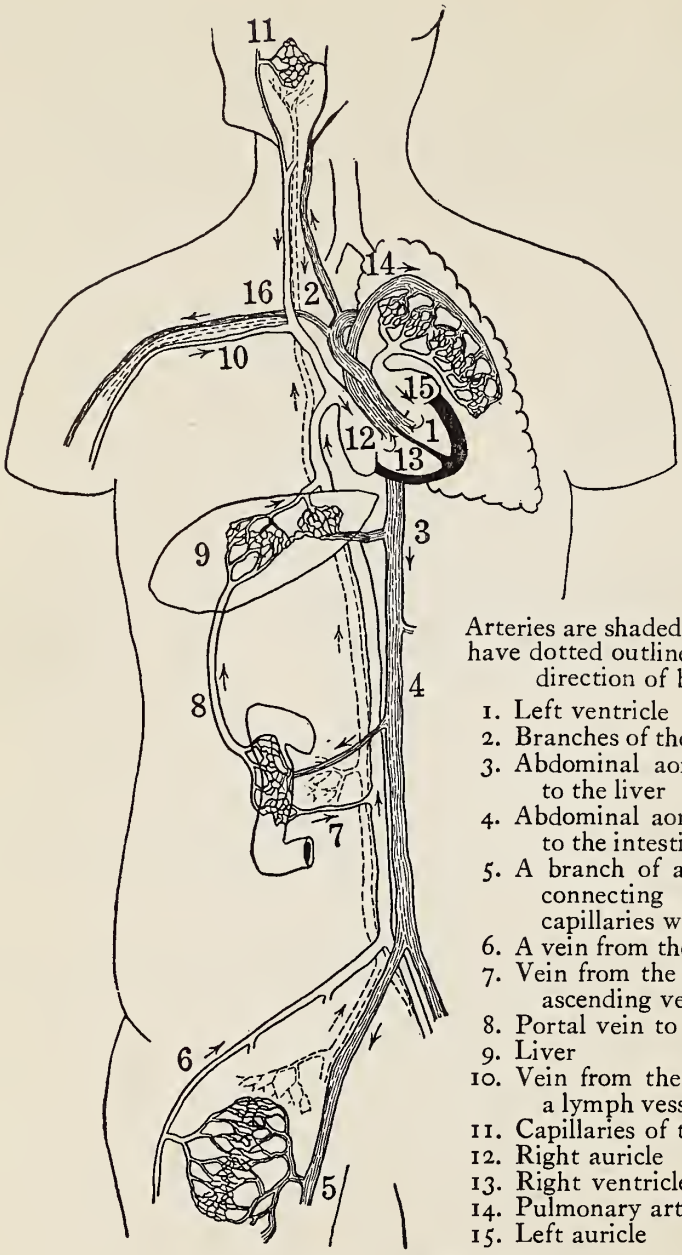


Fig. 94. The heart: exterior and interior

1. Right auricle
2. Left auricle
3. Right ventricle
4. Left ventricle
5. Descending vena cava
6. Ascending vena cava
7. Aorta
8. Pulmonary artery
9–10. Valves between auricles and ventricles

and that the valves in the heart known to the ancients were so placed as to permit the blood to move through the veins toward the heart and through the arteries away from the heart (Fig. 95), Demonstration 18, Appendix, p. 304.

He demonstrated that it was possible for the blood to move from the heart through the lungs and back to the heart. To show this he tied one end of a small open tube into a bladder filled with



Arteries are shaded; lymph vessels have dotted outlines; arrows show direction of blood flow

1. Left ventricle
2. Branches of the aorta
3. Abdominal aorta and branch to the liver
4. Abdominal aorta with branch to the intestine
5. A branch of aorta to the leg connecting through the capillaries with
6. A vein from the leg
7. Vein from the intestine to the ascending vena cava
8. Portal vein to the liver
9. Liver
10. Vein from the arm; above it a lymph vessel from the arm
11. Capillaries of the mouth
12. Right auricle
13. Right ventricle
14. Pulmonary artery to the lung
15. Left auricle

Fig. 95. Course of circulation

water. The other end of the tube was tied into the pulmonary artery where it leaves the heart. By squeezing the bladder he forced the water along the artery to the lungs and soon this water appeared flowing from the pulmonary vein into the left auricle, cut open so it could escape. Strange he did not demonstrate the circulation through the body in a similar way. He concluded, however, that since he had shown that it was possible for the blood to flow through the lungs it must similarly be possible for the blood to circulate from the left ventricle through the body and back to the right auricle (Fig. 95). Demonstration 19, Appendix, p. 305.

Harvey, like Vesalius, suffered for his doubt of the old venerated authorities, Galen and Hippocrates. Many men thought he was a lunatic and his patients refused to be treated by one who disbelieved in the good old ideas. Most physicians were his opponents. Fortunately the king believed in him and retained him as his physician. When the battle of Edgehill was about to begin, King Charles gave the young princes, the Duke of York and the Prince of Wales, to Harvey for safe keeping. He trusted him, not only as a physician, but as a discreet and honorable man. Harvey took the royal lads off to a safe distance and while they watched the battle in which the king defeated Oliver Cromwell, he sat and read the medical book he had brought in his pocket.

CHAPTER VIII

MALPIGHI AND HIS TIMES

MALPIGHI DISCOVERS THE CAPILLARIES

HARVEY DID not know how the blood went from the ends of the small arteries to the little veins. He thought it flowed out from their open ends into the tissues and then was taken in by the open ends of the small veins. It remained for Marcello Malpighi (Fig. 96) to discover the capillaries. In a letter to his old teacher, Borelli, in 1661, he writes:

I might have believed that the blood itself escaped into an empty space and was gathered up again by a gaping vessel and by the structure of the walls. But an objection to this view was afforded by the movement of the blood being tortuous and scattered in different directions and by its being united again in a determinate part. My doubt was changed into certainty by the dried lung of the frog which to a very marked extent had preserved the redness of the blood in very minute tracts (which were afterwards found to be vessels) where by the help of our more perfect glass there met the eye no longer scattered points . . . but vessels joined together in a ring-like fashion. . . . Hence it was clear to the senses that the blood flowed away along tortuous

vessels and was not poured into spaces, but was always contained within tubules, and that its dispersion is due to the multiple winding of the vessels.

(Demonstration 20, Appendix, p.) 306.

THE EDUCATION OF MALPIGHI

MARCELLO MALPIGHI was one of the most able of these men who were busy finding out new things about the structures and activities of the parts of the human body. He was born at the little town of Crevalcore near Bologna, Italy, in the year 1628. His parents were well-to-do farmers and he was the oldest of eight children. His father and mother decided to give this oldest son a good education. So Marcello went to school to prepare for the university and entered as a student in philosophy at the University of Bologna in 1645. Four years later his mother and father both died within a few weeks of each other. So Malpighi was forced to leave school to attend to the business affairs of the family. It was not until 1651 that he could resume his studies. And now he decided

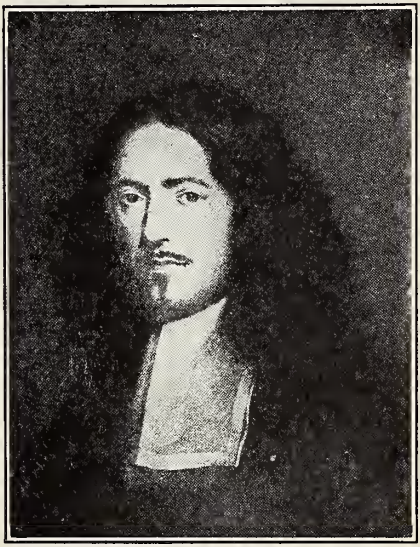


Fig. 96. MARCELLO MALPIGHI [Locy]

to become a doctor. Two years later he took his degree both in philosophy and in medicine and began practice as a doctor in Bologna. The next year he married Francisca Massari, sister of the Professor of Medicine at Bologna. In 1656 he was appointed professor of medicine, for Massari had died the preceding year. He was called at once, however, to a better position at the University of Pisa, where he was professor of Theoretical Medicine, or physiology, for three years. Then he came back to Bologna as Pisa did not agree with his health. Once more, in 1662, he moved, this time to the University of Messina. But the University of Bologna recalled him in 1666 and here he remained in the Chair of Medicine for twenty-five years.

THE UNIVERSITY OF BOLOGNA

BEFORE proceeding farther to tell of his discoveries, let us try to catch the flavor of his times and his surroundings. It will help us to form a picture of the conditions under which not only Malpighi worked but others also of these men whose labors we are trying to appreciate.

The University of Bologna where Malpighi was a student and where he spent so many years teaching is the oldest in Europe. The Universities of Paris in France, and Oxford in England, are almost as old. It was founded certainly as early as the eleventh century, possibly much earlier. It was

therefore a school with a history of some six hundred years when Malpighi came to it to study. At first it was not called a university but was just a group of students, a guild, banded together to employ teachers to instruct them. The students elected some of their own number as "rectors" to hire the professors and to arrange the pay, to govern the student body and to settle disputes that were likely to arise with the townspeople. In these early days there was no university building, but the teachers met their students in their own homes or in rooms hired for the purpose. The students fined their professors if they failed to meet their classes and if a teacher wanted to leave town for a weekend he must deposit a sum of money with his rector to make sure he would be back on time to do his teaching. When the fame of these teachers had spread so that students flocked to Bologna from all the neighboring countries the school was called a *Studium Generale*. It was not until about the fifteenth century that it became known as a university.

Now most of these curious customs had disappeared by Malpighi's time, yet enough of them remained to make the University of Bologna in which he studied and taught quite unlike the ones with which we are familiar in the United States. In his day the students still elected their rector but the professors were employed and paid by the university. The university was now housed

in an adequate building. This was completed in 1562; it was and still is known as the Archiginnasio. Finally this building became too small and about a hundred and twenty-five years ago the university moved to more commodious quarters. The medical students occupied certain classrooms on the front of the second floor of the building and other rooms about the central court. There was also a fine anatomical theater built in 1595 and enlarged in 1637. Its walls were of wood, finely carved, with statues of famous anatomists and physicians and, in Malpighi's time, a beautiful ceiling of cedar wood was built in. Undoubtedly he attended some classes as a student in this theater and later taught here as a professor.

THE CITY OF BOLOGNA

Much older, however, than the university is the city of Bologna itself. On the fertile plain it occupies at the foot of the mountains between the rivers Reno and Savena, there lived in prehistoric times a people called the Ligurians, whose stone implements are still frequently found in excavations. These people were conquered by the Umbrians, who founded a city on the site of Bologna which they called Felsina. It was a city of importance by the time of the fall of Grecian Troy, made famous in Homer's poetry. These Umbrians were lords of all central Italy and Felsina was their

capital. Bronze and brass utensils, finely sculptured, pottery and other evidences of these Umbrians are unearthed when foundations are laid for new buildings in Bologna. In the early part of the fifth century B.C. the Umbrians were in turn conquered and absorbed by the Etruscans, who came from Asia Minor. Bits of the walls of old Etruscan buildings made of stones nicely fitted together without mortar are still to be found in Bologna. In 389 B.C. the Etruscans were conquered by a tribe of the Gauls, the Boi, who renamed the city Felsina, Bononia. They in turn were made subjects of the Romans, 198 B.C., and the city and adjacent country became a Roman colony. The city was again devastated by the Huns and Vandals about A.D. 400. Next it was subjugated by the Lombards, invaders from the Scandinavian peninsula, and became part of the Kingdom of Lombardy. But the Franks, under King Pippin and his son, Charles the Great, soon overcame the Lombards, and Benonia, now renamed Bologna, was made part of the Kingdom of the Franks. In A.D. 966 the city was granted its freedom and became one of the several powerful free cities of Italy. Its career was a stormy one through many following centuries. Sometimes it was a dependency of the Pope, sometimes a dependency of some adjacent kingdom that conquered it, sometimes it was ruled by one or another of its own powerful and wealthy nobles.

BOLOGNA AS MALPIGHI SAW IT

The streets along which Malpighi walked and the old buildings that lined them would speak to him of this long and varied history of his beloved city. The central part of modern Bologna, with its streets at right angles, is the oldest part. From its margin the streets radiate to the gates of the more

Fig. 97. Ancient Bologna and its Wall [James]

recent city. The old city was surrounded by an ancient wall (Fig. 97). New walls were built as the place grew and the most recent one still stands partially intact. There are also fragments of the older walls and gates which were more numerous and more complete in Malpighi's time. Many old buildings still stand, some of them in much the

same condition as when he saw them. The church of Saints Peter and Paul was first built in the third century A.D. It was repeatedly destroyed and rebuilt as the city was subject to conquest after conquest. It stands now about as rebuilt in 1019. Other churches have been built close around it, forming a group of seven now known as the

Fig. 98. Palazzo Commale, Bologna, Italy

St. Stephano group. The church of Saints Peter and Paul was for centuries used for the university functions when degrees were granted. Probably here Malpighi received his *laure dottorale* and likely attended many such graduation exercises here during his career as professor.

The Palazzo del Podesta, built in 1121, the Palazzo Publico begun in 1287, the Mercanzia

completed in 1382, the famous leaning towers built about 1110 (Fig. 99), and many other buildings both public and private, including homes of some of the wealthy noble families stand in the old city much as they were in Malpighi's day. The university building itself is in the old city. It has been the habit for centuries to build in connection with buildings arched arcades over the sidewalks as a protection from rain and the hot summer sun. These still exist so that probably many of the streets look much as they did when Malpighi walked along them to his classes (Fig. 98). He lived in the western district of the city near the Porta Nuovo (the new gate) where most of the "medics" lived. A street in this section is still named Piazzo Malpighi. The law students lived in the southern district of the old city near the Porta Procola.

He must often have been thrilled as looked upon these very ancient buildings and realized what a long and eventful career his city had experienced. Right here where he lived man had been struggling up from the savagery of the Stone Age, climbing up to better things in spite of hardships, through early civilizations that were often temporarily destroyed, yet ever reappeared. So keen and sensitive a man as Malpighi, realizing all this, must have been inspired to play nobly his part in the betterment of mankind. And he did. His were wonderful discoveries.

OTHER UNIVERSITIES

THE University of Bologna at the height of its glory had ten, perhaps twenty thousand students, drawn by the fame of its teachers. The attendance was much less while Malpighi was there. Many students, dissatisfied with the restrictions put upon them by the citizens of Bologna, and having promise of more freedom in other cities, had at various times left Bologna in a body and gone to other nearby cities to start universities. Thus was Bologna the mother of many other Italian universities, notably that of Padua, which became her great rival, and was in Malpighi's day more noted, and attended by many more students.

One wonders that these famous schools had so many students, especially when one recalls that other equally great universities existed in France, Germany, England, and other countries. But a university education was the one means of entrance to the learned professions, to the important and very numerous clerical appointments in the gift of the church and to most political appointments.

MALPIGHI'S WORK

THE pictures of Malpighi show a face that indicates strength of character, refinement, and intellectual penetration. He was much beloved by his friends and by his students. He treated even his enemies with rare consideration. He was never

physically robust and yet he accomplished an amazing amount of work. His discovery of the capillaries has already been noted.

While teaching at Messina, a city on the seashore, Malpighi took the opportunity to study the structure of fishes and of still simpler sea animals to be had there in such abundance. He came to the conclusion that the organs and their actions in these simple creatures corresponded very closely to similar organs and functions in man. So he decided a doctor could well study these simpler animals and find out much about the way man's organs were built and worked. Such animals were much more easily obtained and dissected. Malpighi thus became the first real comparative anatomist.

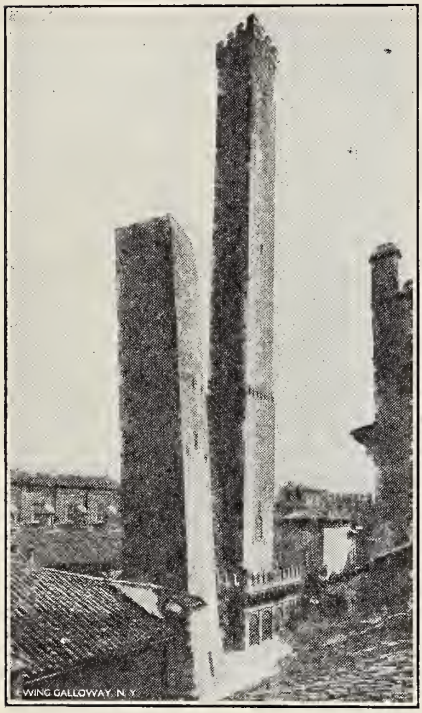


Fig. 99. Leaning Towers of Bologna

As he studied the lungs in frogs and newts (Fig. 100) he saw that the windpipe divides repeatedly on entering the lung, and that its tiny branches lead to air spaces surrounded by the thin

tissue of the lung (Demonstration 21, Appendix, p. 307). It was in this tissue he saw the tiny capillaries carrying the blood and therefore drew the conclusion that not only in frogs and newts, but in the higher animals including man, the lung is an organ whose work is to bring the air and the blood close together. The real meaning of this had to await later discoveries, but Malpighi had taken a long step in understanding the work of the lungs.

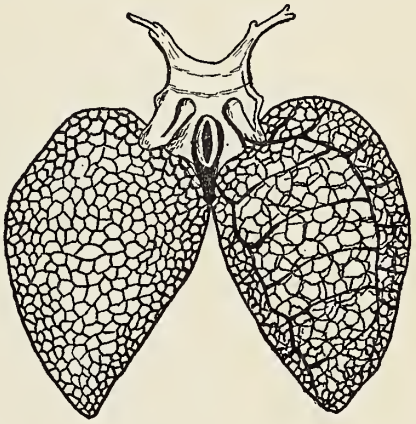


Fig. 100. Lungs of Frog [After Malpighi's drawing in the possession of the English Royal Society]
The windpipe is slit open. The lung at the left of the figure shows the numerous small very thin-walled air sacs of which it is largely made. The right of the figure shows some of the blood vessels which lead to the capillaries in the transparent walls of the air sacs

In that century in which Malpighi lived, many men aided in the discovery of the ducts or outlets of the glands. Wirsung, professor of anatomy at the University of Padua, had found in 1642 the duct of the pancreas. Wharton and Stenson, soon after, found the ducts of the salivary glands and Stenson the duct of the tear gland. Malpighi showed that the liver was also a gland secreting bile and that the bile duct is the vessel that carries the bile out of the liver into the intestine. This discovery

again was made by first studying the liver in simpler animals like snails and fishes. But what is most remarkable was that he proved his point by experiment. He drained the gall bladder and the bile duct of a kitten. Then after tying the bile duct close to the intestine so no fluid could get into it from the intestine he observed that the duct and gall bladder soon filled up again, the bile evidently coming from the only possible source, the liver. It was such testing of his ideas by experiment that marks Malpighi as a man who was far in advance of his times, an exponent of the modern ideas of scientific procedure. He also added much to our knowledge of the way the kidneys work. He discovered the taste organs in the tongue and the sensitive papillæ in the skin which are so serviceable in our sense of touch (Fig. 101).

He was also using the newly discovered microscope both on the tissues of animals and on those of plants. He wrote a remarkable book, illustrated with many drawings, on the structure of the silk worm. Another book was written on the structures of plants, also fully illustrated. In this he shows the structure of bark, wood, roots, etc., even giving their cellular nature. Thus he began the study of histology.

He studied the development of the chick from the egg. His treatise on this subject, full of careful drawings, was by far the best that had been done

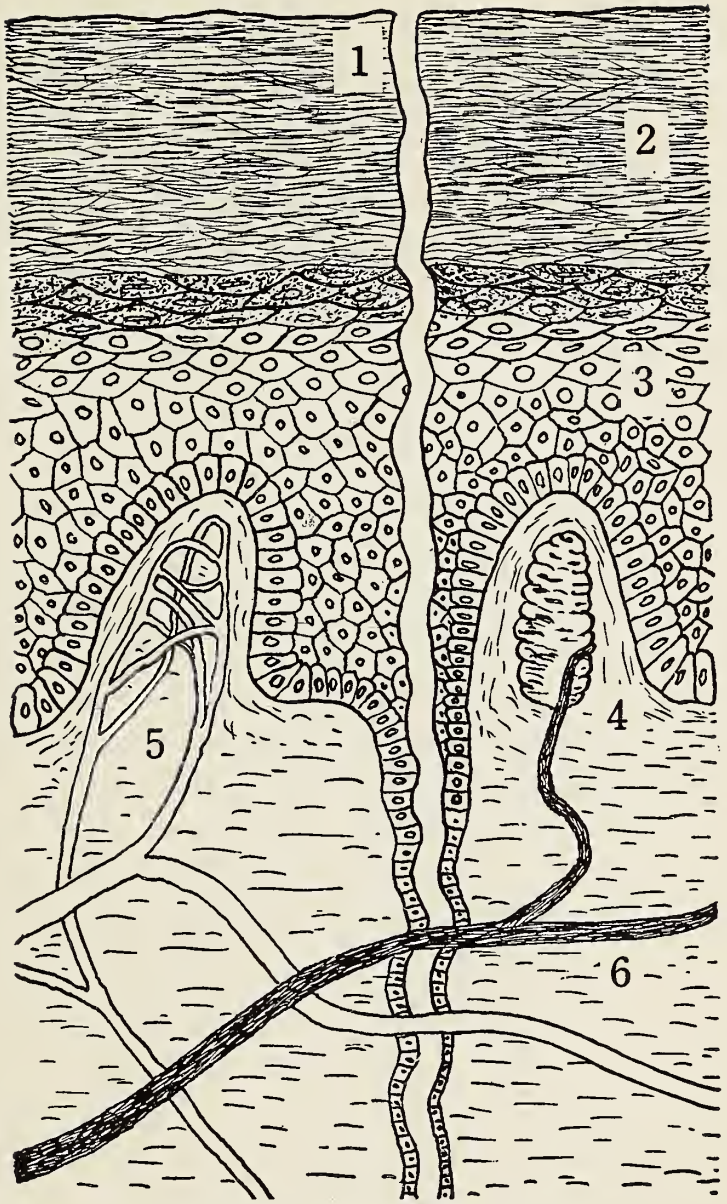


Fig. 101. Diagram of section of the skin

1. Duct of a sweat gland.
2. Horny layer of epidermis.
3. Cellular layer of epidermis.
4. Papilla of the dermis with nerve ending in a touch corpuscle.
5. Papilla with loop of capillaries.
6. Connective tissue of dermis.

and it was the standard work on the subject for nearly two hundred years. So he also laid the foundations of embryology.

We marvel that one man, always rather sickly, could do so much. He was practicing during all these years as an eminently successful physician, teaching at the University and yet finding time for his investigations. He spent his winters in his house in Bologna, but during the summers he lived at a small country place in the neighboring town of Corticella. As he grew older, more and more of his time was spent in this delightful place. Toward the close of his life, 1691, he was summoned to Rome to be the private physician of the Pope. But he lived there only three years. His faithful wife died only a few months before Malpighi suffered a fatal stroke of apoplexy. His body was brought back to Bologna and buried with great honor.

Let us recall again the old explanations of the way in which the body works. Men could readily see that there were several things that needed explanation even from the early times when they first began to think about these problems. The body grew from birth until the individual attained full size. It was able to heal cuts and other wounds. The body during life was always warm, it was able to move and do things in an intelligent and purposeful way. Men knew that the food was taken up from the stomach and intestines and

that this absorbed material went to the liver. Here, they thought, it was endowed with a nutritive property. They did not know just how this came about nor just what this property was, so they called it "natural spirits." The blood containing these "spirits" went to the heart and in the left side of the heart the blood was mixed with air that came from the lungs and in some unknown way the "natural spirits" produced the "vital spirits," that made movements possible. In due time the blood with "vital spirits" in it reached the brain and was then endowed with the power of control, or the "animal spirits."

The heart was the organ that generated heat, just how was not known. The air that came to the heart from the lungs not only helped in producing the "vital spirits" but it also helped to maintain the heat, and at the same time to keep the heart from getting too warm. In its work the heart also produced certain harmful vapors which were carried back to the lungs by that same vessel, the pulmonary artery, which brought the air to the heart from the lungs, but these vapors were carried in the opposite direction, namely, to the lungs.

Now the work of Harvey had shown that this pulmonary artery carried blood always in one direction, from the heart to the lungs. His studies had also shown that all parts of the body had blood circulating through them in tiny blood

vessels. Malpighi showed that the lungs consisted of many tiny air sacks and that the walls of these air sacks were supplied with blood vessels. Thus the way was prepared for a truer notion of the nature of breathing and of the way the blood supplies nutrition to the body.

CHAPTER IX

RIVAL PHYSICAL AND CHEMICAL THEORIES

BORELLI

THESE EARLY investigators could not clear up at once these problems of how the body is nourished, how it is kept warm, how it moves in an intelligently controlled way. It took the observations and experiments of many men through hundreds of years to reach our modern ideas in regard to these matters. Perhaps some of these early students saw these problems, but probably most of them only saw the small details on which they were working and did not see the large problem of which they formed a part. Physicians and professors all over the world were working, each trying to clear up some little difficulty and in due course of time these results fitted together and made clear the large problems.

There was one man who believed that these perplexing questions as to how the body worked could all be understood if one just knew the

mechanics that were involved. This man was Giovanni Borelli (Fig. 102). He was born at Naples, Italy, in 1608, in the New Castle, where his father was a soldier. He went to Rome to study mathematics and in 1640 was appointed to the chair of mathematics in the University of Messina. He became very much interested in the work of Galileo and was for a time a pupil of his. Galileo had taught both at Pisa and at Padua. Through his work, and that of other men, the idea was becoming firmly established that many of the things in nature could be explained through an understanding of certain laws of mechanics which they were discovering experimentally. Borelli, with his skill in mathematics, was much taken with these newly discovered physical laws, most of which could be expressed in mathematical form. In 1656, Borelli left the University of Messina and went to take the chair of mathematics in the University of Pisa, where he remained for twelve years. During three of these years Malpighi was also at Pisa and these two men became fast friends.

Fig. 102. Giovanni Borelli [Garrison]

It was his contact with Malpighi that interested Borelli in the problems of the human body. He saw that the bones of the body were really levers that were worked by the muscles, and he calculated mathematically just how they worked. He wrote a book on the motions of animals explaining them in terms of simple machines. He knew that when a muscle contracts it swells up in the middle and the two ends are brought nearer each other (Fig. 116) (Demonstration 22, Appendix, p. 307). It was this contraction of the muscle working on the bones in a perfectly mechanical way that produced movements. He thought some sort of fermentation went on in the muscle stimulated by the nerve and that this produced the contraction. This conception was wrong, but his explanation of the mechanics of motion was quite right.

MECHANICS OF THE CIRCULATION

He also undertook to explain in a mechanical way the circulation of the blood. He tried to calculate how much force the heart would have to exert in driving the blood around the body. He saw, too, that the arteries were elastic tubes and that it was the pressure of these elastic tubes on the blood, driven into the arteries by the contraction of the heart, that kept the blood flowing in a steady stream through the small blood vessels. He studied the mechanics of breathing and saw

that the air is not drawn into the lungs but enters by its own pressure when the chest is enlarged. He thought, too, that this inhaled air is the chief cause of the life of animals. He learned this through the experiments of Boyle and other English investigators whose work will be described shortly. He was so taken up with his mechanical explanation of the activities of animals that he thought that this process of expanding and contracting the lungs imparted an oscillation to the particles of the blood and that this motion regulated the actions of animals.

MECHANICS OF DIGESTION

Borelli also thought of digestion as largely a mechanical process. He tells how he made turkeys swallow glass globules, small wooden blocks, filberts whose shells are so hard the teeth of a man can hardly crack them. Then, when he killed the turkeys next day and opened them, he found all these things ground up to a fine powder. He did admit that certain birds and fishes that have no teeth and whose stomachs are made of only a thin membrane digest their food by means of a powerful ferment which acts as do certain corrosive fluids on metals. But he thought these were the rare exceptions and that in most animals food was ground up as the main operation in digestion. Borelli's influence lived for a long time after his death and his followers tried to explain

all the actions of the body in a purely mechanical way.

BORELLI'S CLOSING YEARS

THE last few years of Borelli's life were not pleasant. He grew to be a crabbed man, hot tempered, insolent to his opponents and spiteful even with his old friends. In 1668 he left Pisa and went back to Messina. His quarrels with his associates were becoming very bitter, undermining his health and his happiness. Nor did he stay long at Messina. He became involved in a political conspiracy and had to flee. He went to Rome, where for a few years he was supported by Christina, the daughter of Gustavus Adolphus, King of Sweden. She had been queen, after her father's death, but disliking the duties and restraints which the office imposed upon her, she had gone to live in Rome. Borelli was her doctor and also lectured before the group of scientific men she patronized. Finally support from her failed. Borelli's scanty supply of money was stolen by a servant whom he had trusted, and he was forced to join a monastic order. During these years, however, he was busy writing his books. His major book, on the motions of animals, was not published until a year after his death, which occurred in 1679. He was intellectually a great man though not a lovable one. His last years were saddened by the lack of friends, and by the

bitter attacks of enemies whom he had made largely by his own ugly manners.

VAN HELMONT AND THE CHEMISTRY OF THE BODY

But now there was growing another school of thinkers and experimenters opposed to Borelli and his followers. They thought that the activities of the body were to be explained in terms of chemical processes, instead of mechanical ones. Perhaps the greatest of these men was Jean Baptiste van Helmont (Fig. 103). He was born at Brussels, in Belgium, in 1757. He studied at the University of Louvain. After trying philosophy and law, with no satisfaction, he began to study medicine. He took his degree at twenty-two years of age, and then spent several years in travel and study. Shortly after his return he married a rich lady and settled down at Vilvorde, a small town near Brussels, and began his investigations. He discovered the gas we now call "carbon dioxide" or "carbonic acid gas," and so was the first to begin the study of the various gases so important

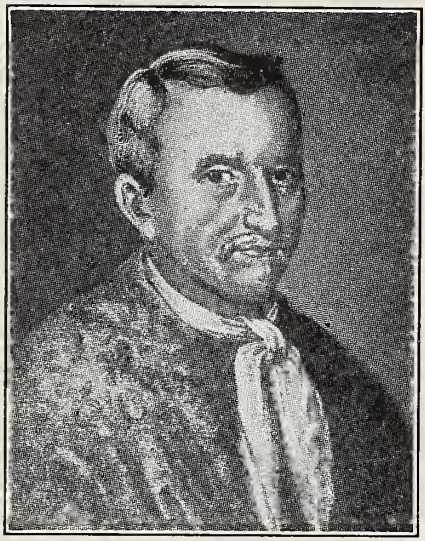


Fig. 103. Jean Baptiste van Helmont [Garrison]

in understanding the activities of the body. He thought digestion was a sort of fermentation, that went on not only in the stomach and intestines, but also in the blood, and, most remarkable of all, in the tissues themselves. He held that each organ and each tissue prepares food for itself by means of a ferment peculiar to it.

SYLVIUS

Fig. 104. FRANCESCO SYLVIUS [Garrison]

THIS idea that the processes of the body were chemical in character was farther advanced by François DuBois, also known as Francesco Sylvius (Fig. 104). It was quite common in those days for men to have two forms for their names, one, in this case the latter one, the Italian equivalent of the other. Sylvius was born in 1614. He took his degree in medicine at Basel, Switzerland, and in 1658 became professor of medicine at Leyden, Holland, which position he held until his death in 1672. Now van Helmont had thought that these fermentive changes that went on in the body were peculiar to living things. But Sylvius declared they were just ordinary processes, the same sort of chemical changes that would go on any-

where (Demonstration 23, Appendix, p. 307). In fact he held that all the chemical changes going on in the body, digestive and all the rest, were just like those going on outside of the body, even in non-living matter. He had almost no facts to base this statement on. It was really a shrewd guess such as a keen mind sometimes makes.

You will recall that about this time the ducts of several glands connected with the digestive tube had been discovered. Men wondered if they could detect in the secretions from these glands any signs of the way in which they brought about digestion. They had the notion, however, that chemical changes were due to differences in the behavior of acids and alkalis and so at first their interest was chiefly in finding whether these fluids from the glands were acid or alkaline (Demonstration 24, Appendix, p. 308).

Hermann Boerhaave, born four years before Sylvius died, also came to occupy the chair of medicine at Leyden (1701–38). He held that the juices of these glands were the agents of digestion but claimed that the process was not one of fermentation but of mere solution.

While some of these investigators, notably de Graaf, had collected the secretions of one or two digestive glands, it had been found difficult to collect these juices in anything like pure form. And their action on foods in the digestive canal had to be guessed from the condition of the food

observed when animals were killed and opened for study. Now, however, there began some ingenious experimental work.

RÉAUMUR AND HIS KITE

A Frenchman named René de Réaumur published in the Memoirs of the Academy of Science of Paris a paper on the digestive process in birds (1752). Some birds like owls and kites eat the animals on which they feed, hair, feathers, bones, and all, and then after digesting all the parts that can be so digested, the rest of the material is thrown up out of the mouth. Réaumur gave a kite small metal tubes filled with meat. The ends of these tubes were covered with wire netting. These the kite obligingly swallowed, evidently relishing the smell of the contained meat. When the tube was later thrown up, the meat was almost or completely gone, dissolved apparently by the digestive juices. Even bits of bone were largely dissolved. If any of the meat was left in the tube, it showed no signs of rotting. Often the tubes when ejected contained more or less of a bitter yellowish fluid which Réaumur thought was the digestive fluid. If the tubes were filled with vegetables or grains, such material was not dissolved, so apparently this digestive juice acted only on certain sorts of substances.

Next Réaumur give the kite tubes containing

pieces of sponge. When these were ejected, he could squeeze from the sponge quite a quantity of the digestive juice. He then tried to dissolve bits of meat in this juice contained in small vessels outside the body, also putting similar bits of meat into water in other vessels. All these he kept about at body temperature. The meat in the digestive fluid did dissolve slightly and it did not putrefy nor rot, as did that in the water. The latter showed no sign of digestion. So he concluded that digestion is a process of solution due to the digestive fluids secreted by the glands and that it is not a process of fermentation like putrefaction or decay.

SPALLANZANI AGAIN

THAT same Lazaro Spallanzani, to some of whose experimental work we have already referred (see Fig. 7, p. 10) tried Réaumur's method on himself. He was a professor at this time in the University of Pavia. He used metal tubes similar to Réaumur's, except that he made holes in the tubes themselves so that the digestive fluids could get to the contents more readily. He not only experimented on animals as Réaumur had done, but he himself swallowed linen bags containing meat, bread, and other foods. He also swallowed small wooden tubes with foods in them, the tube perforated so the digestive juices could easily enter.

He also tried swallowing similar tubes in which bits of sponge were used to absorb the digestive fluids. He made himself vomit in order to collect samples of the digestive fluids, but found the process was so disagreeable that he gave it up. It was a rather courageous thing to try, this process of swallowing metal and wooden tubes, for he was not sure what effect they might have on himself. Of course, he had experimented on animals and knew that in most cases they survived, but that gave no absolute assurance that the human digestive system could stand such treatment. However, he did live to write the results of his experiments and for many years afterwards. He also tried letting animals swallow pieces of meat and other foods tied on the end of a string. After these had been in the animal's stomach for some time, he pulled them out to see what digestive changes had occurred. You see he was an exceedingly inquisitive man and very ingenious in devising experiments as well as skillful in conducting them.

As a result of all these experiments, he came to the conclusion that in all animals the food is dissolved and that the mechanical grinding of the food by the teeth or by the gizzard in birds is merely a preparation for the solution. He also concluded that digestion is not a putrefactive process; in fact, the digestive fluids seem to prevent rotting.

WILLIAM BEAUMONT SEES INSIDE THE STOMACH

In 1825 a surgeon of the United States Army, William Beaumont, stationed at the fort on the Island of Mackinaw, Michigan, had a remarkable opportunity to study the digestive processes. An Indian half-breed, Alexis St. Martin, met with an accident. A gun, unexpectedly discharged, tore away the front of his body and a part of the outer wall of his stomach. Under the care of Beaumont the wound healed but left a small opening so one could see into the stomach. Beaumont could watch the movements of the stomach as the food was churned about in it. He could see that the gastric juice was secreted only in the presence of food and he thought this gastric juice must contain some active substance that caused the digestive changes. He could also see how long it took various foods to digest and how completely they were dissolved in the stomach.

PEPSIN IS DISCOVERED

In 1835 a German professor, Theodor Schwann, separated out from the gastric juice a substance which he called pepsin. He proved by experiment that this would change or digest certain food materials so that when in solution they could pass through the walls of the stomach and the intestine (Demonstration 25, Appendix, p. 309). Later another investigator (1845, Louis Mialhe) found

in the saliva a similar substance that can change starch into sugar, and later on other like substances were found in the digestive fluids of the intestine.

So it gradually became clear that digestion is due largely to the work of enzymes, as these substances are called, which merely by their presence change insoluble food materials into soluble ones that can pass out of the digestive tract into the blood.

WÖHLER MAKES AN ORGANIC SUBSTANCE

In 1824, Friedrich Wöhler, working under the great German chemist, Liebig, found that certain relatively simple substances in the body combine into more complex compounds. Up to this time it was thought that animals must take their foods already formed by plants and that plants alone have the power to combine the simpler substances into more complex ones which manifest the property we call life. This same Wöhler also made for the first time in his laboratory a substance that had been obtained heretofore only from living things. Men had thought that the products obtained from plants and animals could be produced only by such living things. Wöhler proved that this is not so. Now a great many plant and animal products are manufactured in the chemist's laboratory. So it was shown that in all probabil-

ity the chemical processes going on in the body of the animal or the plant were like those found anywhere else and that if one could follow these processes, he would understand in large measure how the body works.

CHAPTER X

NEW LIGHT ON RESPIRATION

ROBERT BOYLE

Now let us go back and trace the increasing knowledge regarding the nature of respiration. You will remember that Borelli had concluded that breathing is the chief cause of the life of animals. That conclusion was partially due to his own work, but largely to some experiments with which he was familiar, experiments that had been done in England by Robert Boyle (Fig. 105). Boyle was an Irish gentleman of considerable means who devoted much of his time to scientific work. He devised a successful air pump by means of which the air could be fairly well removed from a glass vessel. He observed that a candle put into such a vessel went out as the air was exhausted, apparently unable to burn in the partial vacuum. He also observed that an animal put into such a glass vessel died as the air was exhausted. If he put an animal like a mouse and a lighted candle also into the glass vessel and then

exhausted the air, the animal died and the candle went out more quickly than when either one was present alone. He thought, therefore, that both candle and animal required air, the one for burning, the other for respiration; and since both were using the air, it was exhausted more quickly when both were present.

Fig. 105. ROBERT BOYLE

Boyle's workshop or laboratory at Oxford was a meeting place for many of the people who were interested in science. He, himself, was familiar with many phases of science and experimented in several fields. He is most famous, perhaps, for his discovery of what is still known as Boyle's Law, which expresses the relation between the volume of a gas and the pressure to which it is subjected. This group of scientists, meeting first in Boyle's rooms at Oxford and later in London, organized a society for the discussion of the scientific discoveries that were coming so rapidly. In 1660, as already noted, this society was chartered by the king, becoming the Royal Society for Improving Natural Knowledge. The society is still one of the foremost scientific organizations in

the world and to be elected a member of the Royal Society is a very distinguished honor.

ROBERT HOOKE

Robert Hooke was a member of this Royal Society. He was for some time assistant to Robert Boyle in his Oxford laboratory. Later he became curator of the Royal Society. He was not only in charge of the apparatus that was collected by the organization but it was his duty to present various experiments at their meetings.

You will remember that Borelli thought that the expansion and contraction of the chest in breathing imparted to the blood particles a vibration which in some way or other generated the motions of the body characteristic of living animals. It was the general notion of his day that the essential part of respiration was this alternate expansion and contraction of the chest. Now Hooke showed at one of the meetings of the Royal Society that this was not true. He opened the chest cavity of an animal, cutting away parts of the chest wall so that the alternate expansion and contraction could not go on, but he kept the lungs inflated with fresh air constantly passing into them through a tube. The animal continued to live, showing that it was the supply of air in the lungs that was essential to life and not the expansion and contraction of the chest in respiration.

RICHARD LOWER

THERE was another man about this same time, Richard Lower, who was also teaching at Oxford and was also engaged in the practice of medicine. He repeated this experiment of Hooke's and noticed another interesting thing—that the blood flowing to the lungs in the pulmonary artery was dark, but that the blood flowing from the lungs back to the heart in the pulmonary vein was bright red. Now it had been thought by the older scientists that this change in the blood occurred in the heart. Lower also noticed that when the experimenter ceased pumping air into the lungs, then the blood going from the lungs to the heart became promptly dark in color. He therefore concluded that this change in color was due to the contact of the blood with the air in the lungs.

JOHN MAYOW AND THE NATURE OF RESPIRATION

THE next man to make an important contribution to this work on the nature of respiration was John Mayow (Fig. 106). His father and mother came to London from Cornwall in the southwestern part of England. John Mayow was born in London in 1643 and was educated at Oxford, being a scholar at Wadham College (Fig. 107). Promising lads were appointed scholars and thereby had part of their expenses paid. The funds for such provision came from moneys left by interested people, and

those lads who obtained their assistance from the same fund lived together in a "college" maintained by this fund.

John Mayow was a lawyer by profession and a noted one, at that, yet he was exceedingly interested in science and was a fellow of the Royal Society. Mayow had experimented with saltpeter, a substance which is used with charcoal and sulphur to make gunpowder. He found that if you mix saltpeter and sulphur together, and then heat them in a closed vessel from which the air has been exhausted, the sulphur will soon catch fire and burn much as it would if it had been touched off in the open air. He reasoned, therefore, that there must be something in saltpeter which is also in the air and that when sulphur or any other substance burns in the air it does so because it unites with this same thing. He weighed certain substances before and after they were burned and he found that they weighed more after they were burned than before, showing that in the process of burning they had absorbed some-

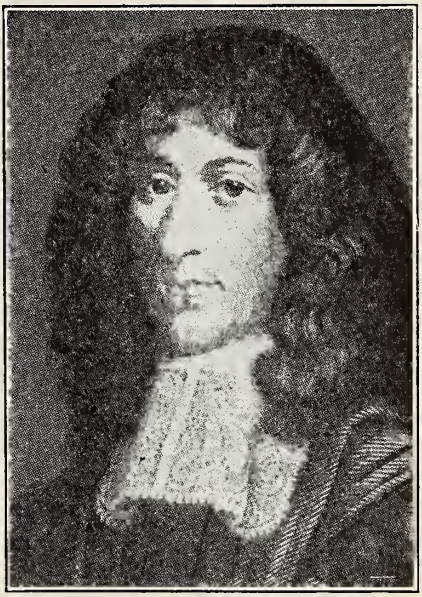


Fig. 106. JOHN MAYOW [Garrison]

thing. Respiration, he therefore concluded, does not use all of the air but only a part of the air. When he burned a candle in a vessel open only at the bottom and set the bottom of the vessel in water, he noticed that as the candle burned the water rose in the vessel. Similarly, if he put a small animal into a vessel over water, the water rose in the vessel as the animal breathed. In the

Fig. 107. Wadham College, Oxford

latter case the animal in time dies while in the former the candle goes out. So Mayow reasoned that respiration and burning were alike in that each used up a part of the air, but only a part of the air since the water rose until it filled but a small part of the vessel (Demonstration 26, Appendix, p. 309).

LAVOISIER REDISCOVERS AND NAMES OXYGEN

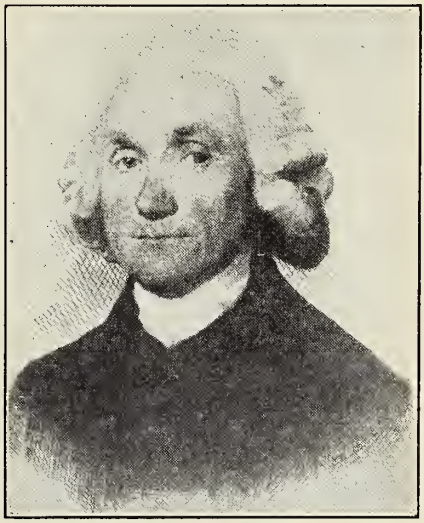


Fig. 108. JOSEPH PRIESTLY [Thorpe]

It was remarkable that these discoveries of Mayow's attracted so little attention. In fact, they were soon forgotten and his work had to be done over again a hundred years later by Joseph Priestley (Fig. 108) in England and by Lavoisier (Fig. 109) in France before it was accepted. Lavoisier named this substance which Mayow really discovered *oxygen*. Priestley was the man who rediscovered it and also showed over again that respiration and burning are similar processes.

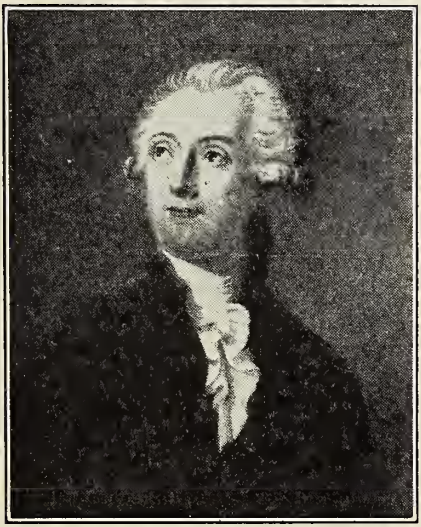


Fig. 109. ANTOINE LAVOISIER [Mayer]

Lavoisier, however, saw things much more clearly than did his predecessors. He showed that the oxygen taken into the body through the lungs is used in burning, and that, as a result, carbon dioxide

is given off just as it is when carbon is burned outside of the body. Furthermore, Lavoisier maintained that, by means of this process of burning which is going on in the body, a temperature is maintained which is considerably above the usual temperature of its surroundings. It remained for later investigators, however, to show how the oxygen taken into the lungs is carried by the red blood corpuscles of the blood to the various tissues and that it is in these working tissues that the burning really goes on (Demonstration 27, Appendix, p. 310).

THE BODY MADE OF CELLS

BACK in 1838 and 1839 Schleiden, a botanist, and that same Schwann who found the pepsin in gastric juice, discovered that the living parts of plants and animals are made up of tiny units called "cells." These are very small bits of the substance known as "protoplasm," which is the only thing that manifests life. This term "protoplasm," first used by Purkinje in 1839, is really the name of a group of substances, somewhat jelly-like in appearance, all closely related, chemically so complex that we do not yet know their exact composition. These cells, all made of this protoplasm, have different forms and function in the various tissues. There are gland cells that secrete various products, like those of the salivary glands. The cells that make up muscles have the

peculiar power of vigorous contraction. Nerve cells can transmit impulses from one part of the body to another, as do the nerves of touch when the impulse is conveyed from the finger tip to the brain (Fig. 110) (Demonstration 28, Appendix, p. 312).

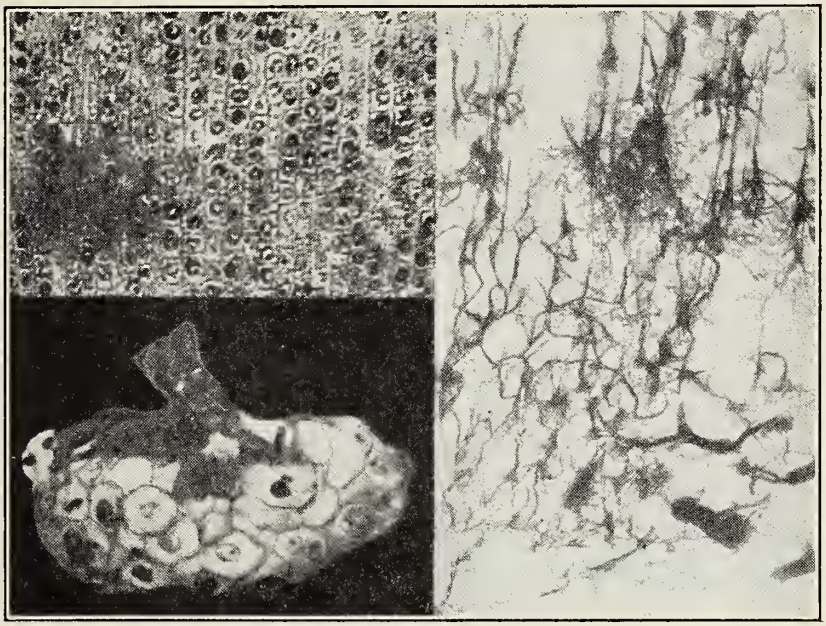


Fig. 110. Cells. Right, brain of cat. Upper left, section of root; lower left, gland of the skin in ear of cat

Gradually it began to be recognized that the various activities manifested by plants and animals, like respiration, motion, secretion, are all due to the activities of these cells. We are made up of these millions of tiny bits of living material, some of which do one thing, some another. The body is really a great society, the cells are the

workers. So it became plain that it is these workmen everywhere in the body that have to be supplied with food and air. The blood needs to circulate to every tiny part of every organ (like a muscle or a gland) that food and oxygen may be carried to these working parts.

THE LAW OF THE CONSERVATION OF ENERGY

Now one final discovery had to be made before all these facts, accumulated through the patient work of so many men, would fit together to make the picture complete. As far as the human body is concerned this discovery was made in 1842 by a German physician, Robert Mayer, who lived at Heilbronn. The discovery was this: The human body uses up, in its activities, a certain amount of energy or power to do work and this energy is supplied by the burning of the material taken in as food. There must therefore be an exact correspondence between the energy taken in, stored in the food, and that used up in the process of living and moving about.

That same year a great physicist, Joule, stated a similar law for the activities of machines and in 1847 Hermann von Helmholtz applied the same idea to all things, living and non-living. The law is now called the law of the conservation of energy. It is stated somewhat as follows: Energy cannot be created or destroyed; it may be transformed from one form to another. This means that if

electricity is produced by a dynamo which is run by an engine burning coal, the mechanical motion of the engine and the dynamo is changed into electricity. And, in turn, the steam generated in the boiler changes the heat from the burning of the coal into mechanical motion. There is always an exact equivalence between the mechanical energy and the heat developed, and an exact equivalence between the mechanical energy and the electricity produced. Food therefore furnishes the material built into the body to produce growth in childhood and also, at all times, it serves as the source of energy or power to do work that enables us to live and move, feel and think.

ENERGY AND FOOD

THE energy contained in food is usually expressed in terms of the Calorie (*C*). The Calorie is the amount of energy in the form of heat which is required to raise one liter of water from 0° to 1° Centigrade. A liter is a little more than a quart. The Centigrade thermometer marks the freezing point 0° instead of 32° as it is marked on the more commonly used Fahrenheit thermometer and the boiling point 100° instead of 212°. Thus 100 degrees on the Centigrade thermometer are the equivalent of 180 degrees on the Fahrenheit thermometer. Since it has been determined by experiment just what the equivalent of this is in terms of the units used to measure mechanical energy, elec-

tricity, etc., it is possible to change the energy value of a known quantity of a food as expressed in Calories into the other units. It makes no difference whether the food be burned inside the body or outside the body, the energy obtained will be necessarily the same. So it is perfectly possible to find the relative value of various foods in terms of their energy content, by burning exact quantities of them outside the body.

The instrument that is used for finding such facts is called a Calorimeter—the name meaning, as you will see, a meter or measure for Calories. The principle of the instrument may be readily understood. Suppose, for instance, a vessel containing 10 liters of pure water were set on a support. Under it there is placed a Bunsen burner or an alcohol lamp. The whole apparatus is enclosed in asbestos covers so that all of the heat that is developed will go into the water and not escape to the surrounding air. A thermometer is put into the water. Now suppose that when the flame burns for ten minutes, the temperature of the water is raised 3°. Evidently, during that time the flame has produced 30 Calories of heat. Next, suppose that a small piece of bread is introduced into the flame. Heat will be produced not only by the flame but the burning of the bread. Let us say it takes the bread ten minutes to burn and that now the thermometer shows a rise of 4° instead of 3°. Forty Calories of energy

have been produced. But, since the flame alone produces 30, the additional 10 Calories must have been produced by the burning of the bread. The actual Calorimeter is a much more carefully constructed instrument than the one described, but its principle of operation is probably understood by the description given.

A great many careful determinations have now been made of the Calorie value of various foods. There is given below a table showing the results of such determinations. (The table is adapted from the American National Red Cross pamphlet "Food"). Today many restaurants show on their menus the Calorie value of the portions of food served so as to enable their patrons to see what they are getting for the money they spend. If one ordered coal and the dealer sent a load of coal, a great deal of which was slate that will not burn, one would refuse to receive it; and yet a great many people buy fuel for their own furnaces, that is, food for the body, without inquiring at all as to whether it has good fuel value or is made up largely of materials that do not burn.

FOOD REQUIREMENTS DURING GROWTH

THE growing child needs food not only to produce energy but also to provide material for growth. More than that, the child is more active than the adult and so requires a larger amount of energy in proportion to his body weight than does the

Name of Food	Approximate Amount of One Serving			Energy Factors—Distribution of Calories		
	Measure	Weight, Ounces	Total Calories	Protein	Fat	Carbohydrate
Milk and Milk Products:						
Milk, whole	½ pt.	8½	170	34	88	48
Buttermilk	½ pt.	8⅘	89	29	12	48
Cheese, American	Cube 1 in.	⅔	89	23	63	3
Cheese, cottage	3⅔ T. (Tablespoon, level)	2	62	49	2	11
Cream, thin	2 T.	1	50	2	43	5
Butter	Pat 1 T.	½	100	1	99	
Ice cream	⅔ C.	5	320	13	202	105
Bread and Cereals:						
Bread, white	1 slice 3×3½×½ in.	⅔	50	7	3	40
Graham bread	1 slice 3¼×2×⅜ in.	½	33	5	2	26
Boston brown bread	½ in. slice, 3 in diam.	1⅕	67	7	6	54
Corn meal, cooked	⅔ cup	6	100	10	5	85
Macaroni, cooked	½ cup	2⅗	50	7	1	42
Oatmeal, cooked	⅔ cup	5¼	67	11	11	45
Rice, white, steamed	¾ cup	4	100	9	1	90
Eggs:						
Egg, whole, in shell	1 egg	2½	70	25	45	
Meat, Fish and Poultry:						
Bacon, cooked	4–5 small slices	½	100	13	87	

Beef, lean, round, broiled	2 slices, $4\times3\times1\frac{1}{8}$ in.	4	200	96	104	
Chicken, roast	1 slice, $4\times2\frac{1}{2}\times\frac{1}{4}$ in.	$1\frac{2}{3}$	100	51	49	
Fish, lean, broiled	1 slice, $4\times2\frac{1}{4}\times1$ in.	4	133	81	52	
Ham, boiled	1 slice, $4\frac{3}{4}\times6\times\frac{1}{8}$ in.	2	150	44	106	
Lamb, chop, broiled	1 chop	$1\frac{3}{5}$	100	40	60	
Liver, calves', broiled	Medium size serving	$2\frac{4}{5}$	100	62	38	
Mutton, roast	1 slice, $3\times3\frac{3}{4}\times\frac{1}{8}$ in.	$1\frac{1}{5}$	100	33	67	
Oysters, raw	$\frac{1}{2}$ cup	$5\frac{2}{5}$	75	37	18	20
Pork chop, lean, broiled	1 chop	$2\frac{2}{5}$	200	64	136	
Vegetables:						
Asparagus	4 stalks 4 in. long	$1\frac{1}{2}$	10	3	1	6
Beans, lima, dried	$\frac{1}{4}$ cup	2	200	42	8	150
Beans, lima, fresh	$\frac{1}{2}$ cup	3	100	23	5	72
Beans, dried	$\frac{1}{4}$ cup	$1\frac{3}{4}$	171	45	8	118
Beans, string, fresh	$\frac{1}{3}$ cup	$1\frac{1}{3}$	15	3	1	11
Beets	$\frac{1}{3}$ cup cubes	2	25	3	1	21
Cabbage, chopped	$\frac{1}{2}$ cup	$1\frac{1}{8}$	10	2	1	7
Carrots, cooked	2 medium	4	40	4	2	34
Cauliflower	$\frac{1}{5}$ small head	$2\frac{1}{3}$	20	5	3	12
Celery	$\frac{1}{4}$ cup	$1\frac{1}{5}$	6	2	.....	4
Corn, fresh	1 ear 6 in.	$4\frac{1}{2}$	50	6	4	40
Corn, canned	$\frac{1}{3}$ cup	$3\frac{3}{5}$	100	11	11	78
Lettuce	$\frac{1}{4}$ head	$2\frac{1}{3}$	12	3	2	7
Onions	3 to 4 medium	$7\frac{1}{5}$	100	13	6	81
Parsnips	$\frac{1}{2}$ cup slices	3	50	5	3	42

Name of Food	Approximate Amount of One Serving			Energy Factors—Distribution of Calories		
	Measure	Weight, Ounces	Total Calories	Protein	Fat	Carbohydrate
Vegetables—*Continued:*						
Peas, dried	$\frac{3}{8}$ cup	$2\frac{4}{5}$	253	70	6	177
Peas, fresh	$\frac{3}{8}$ cup	$1\frac{3}{4}$	50	14	2	34
Potatoes, white, boiled 15 min	1 medium	$3\frac{3}{5}$	100	11	1	88
Potatoes, sweet, baked	1 medium	6	200	12	10	178
Spinach, cooked	$\frac{5}{8}$ cup	$5\frac{1}{4}$	25	3	2	20
Squash, cooked	$\frac{1}{2}$ cup	$3\frac{7}{8}$	55	4	5	46
Tomatoes, fresh	1 small	$3\frac{4}{5}$	25	4	4	17
Turnips, cubes, raw	$\frac{1}{2}$ cup	$2\frac{1}{4}$	25	3	1	21
Fruits, Fresh:						
Apples	$\frac{1}{2}$ large	$3\frac{3}{4}$	50	2	3	45
Banana	1 medium	$4\frac{1}{8}$	75	4	4	67
Cantaloupe	$\frac{1}{2}$ melon	9	50	3	.....	47
Cherries, stoned	$\frac{1}{4}$ cup	$1\frac{1}{8}$	25	1	2	22
Cranberries, fresh	$\frac{1}{2}$ cup	2	25	1	3	21
Grape fruit	$\frac{1}{2}$ large	$7\frac{1}{2}$	100	7	4	89
Grapes, white	22	$3\frac{2}{3}$	100	5	15	80
Huckleberries	$\frac{1}{2}$ cup	$2\frac{1}{3}$	50	2	4	44
Oranges	1 medium	$7\frac{1}{8}$	75	5	2	68

Peaches	3 medium	$10\frac{1}{2}$	100	6	3	91
Pears	2 medium	$6\frac{1}{3}$	100	4	6	90
Pineapple	2 slices 1 in. thick	$8\frac{1}{5}$	100	4	6	90
Plums	3–4 large	$4\frac{2}{5}$	100	5	.....	95
Rhubarb	1 cup	$3\frac{4}{5}$	25	2	7	16
Strawberries	$\frac{2}{3}$ cup	$4\frac{1}{2}$	50	5	7	38
Fruits, Dried:						
Apricots	9 halves	$1\frac{1}{3}$	100	7	3	90
Dates, unstoned	3–4	1	100	2	7	91
Figs	3 large	$2\frac{1}{5}$	200	10	2	188
Prunes	4 medium	$1\frac{2}{5}$	100	3	.....	97
Raisins	$\frac{1}{4}$ cup	1	100	3	9	88
Nuts:						
Peanuts	20–24 single nuts	$\frac{3}{5}$	100	19	63	18
English walnuts	8–16 meats	$\frac{1}{2}$	100	11	82	7
Sugar and Sweets:						
Sugar	1 T.	$\frac{2}{5}$	50	.....	.....	50
Honey	1 T.	1	100	1	.....	99
Maple Syrup	2 T.	$1\frac{3}{5}$	133	.....	.....	133
Molasses	2 T.	$1\frac{3}{5}$	133	4	.....	129
Corn Syrup	$1\frac{3}{4}$ T.	$1\frac{1}{2}$	100	.....	.....	100
Gingerbread, plain	Piece, $2\times1\frac{2}{3}\times2$ in.	$2\frac{1}{5}$	200	14	42	144
Sponge cake, 2 eggs, hot water	Piece, $3\times2\frac{3}{4}\times\frac{7}{8}$ in.	$1\frac{1}{2}$	150	11	10	129

adult. The food requirements of the child, therefore, are not the same as are those for the adult. There is shown in the accompanying graph (Fig. 111) the food requirements of the child.

It will be noticed that there are two lines in the graph, one showing the number of Calories required per day, the other the amount of proteid required

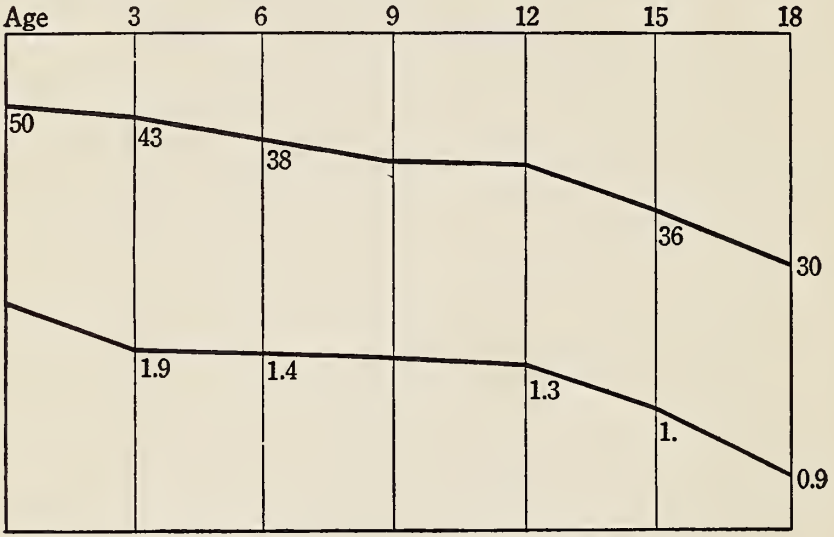


Fig. 111. Graph showing food requirements of the child
Upper line—calories per pound of body weight. Lower line—proteids in grams per pound of body weight

each day, and this item needs some explanation. The living material, the protoplasm of the body, is a protein. It of course wears out as it is active and must be replaced. This proteid contains certain substances, notably nitrogen, not found in such foods as starches and sugars, fats and oils. A certain portion of the food used each day needs,

therefore, to be protein. The table of foods given above shows not only the Calorie value of various items of food but also the proportion of protein, carbohydrates (sugars, starches, etc.), hydrocarbons (fats and oils) in each kind of food. You have probably already learned how to use a table of this sort in order to select your meals wisely. This matter is introduced here merely to explain how a table like the one given was made possible by the many discoveries that have been made, enabling us to understand the way in which the body works.

SUMMARY

As the early part of this book was devoted to the development of one theme—the germ nature of disease—so the latter part has tried to make clear a second idea and its development, namely, the law of the conservation of energy as it applies to the human machine. Like any other machine the body must have energy or power to do work in order to accomplish its tasks. The real workers are the living cells throughout the body. While these cells are very small, as a rule, they are also very numerous. It is estimated that we have about 13,000,000,000 of them in the brain alone. In these cells all over the body the process of oxidation or burning is constantly going on, and as a result energy is developed, some of it appearing in the form of heat, some in muscular activity, some in the processes of secretion in the glands and

some in various other activities. The faster we live, the faster we burn up.

In order that the cells may replenish the material used up in this burning process, they must be supplied with food, and in order to accomplish the burning, they must be supplied with oxygen. So the food taken into the digestive tract is changed, if necessary, by the various enzymes so that it can pass into solution or it is otherwise altered so it can go through the walls of the digestive tract and the tiny capillaries and enter the blood stream. The heart is constantly pumping this blood all over the body. Part of the liquid portion of the blood escapes through the pores in the tiny capillaries and helps form the lymph, which bathes the cells in all the tissues. In the lungs the blood receives a supply of oxygen. This is carried in a loose combination with the coloring material of the red corpuscle. When these red corpuscles enter the tiny capillaries they give up the oxygen supply and it passes through the wall of the capillary out into the lymph and is absorbed as needed by the working cells. Thus these cells are supplied with the necessary food and with oxygen.

As a result of the burning process certain waste materials are formed. The carbon in the working cell combined with oxygen forms carbon dioxide, a gas, which is returned to the blood, carried to the lungs and exhaled in the process of breathing. Hydrogen, another element in the working cells,

when combined with oxygen makes water. This is returned to the blood and passed off from the body in part through the lungs as we breathe, in part by the sweat glands as we perspire and in part by the kidneys. Similarly, other elements combine with oxygen and the products are excreted. There are, therefore, a number of organs in the body that are concerned with this process of getting rid of the products of combustion.

THE SOURCE OF THE ENERGY IN FOOD

But since energy can neither be created nor destroyed, what is the source of energy in the foods we eat? It is the energy of the sunlight stored in our foods which is liberated in our bodies and furnishes us with power to do work. We realize that energy comes from the sun in the form of light and heat. There are other forms of energy in the sunlight which we do not perceive through our senses. The green plants provided with the coloring matter, chlorophyl, and some of the other plants like the brown seaweeds that have an equivalent coloring material, are able to use this energy of the sunlight to build the materials we use as food. These materials are manufactured out of such simple things as carbon dioxide, water, ammonia, and others.

Near the author's office a tall tower is under construction (Fig. 112). It is made of blocks of stone. Each block must be hoisted up onto the

tower and be laid in place by the masons. The engine which is busy all day long raising these blocks of stone is spending work-power upon them and that energy is being stored in the blocks. As each stone arrives at the top of the tower, it contains stored energy. If it should be shoved off the top of the tower, it would strike the ground

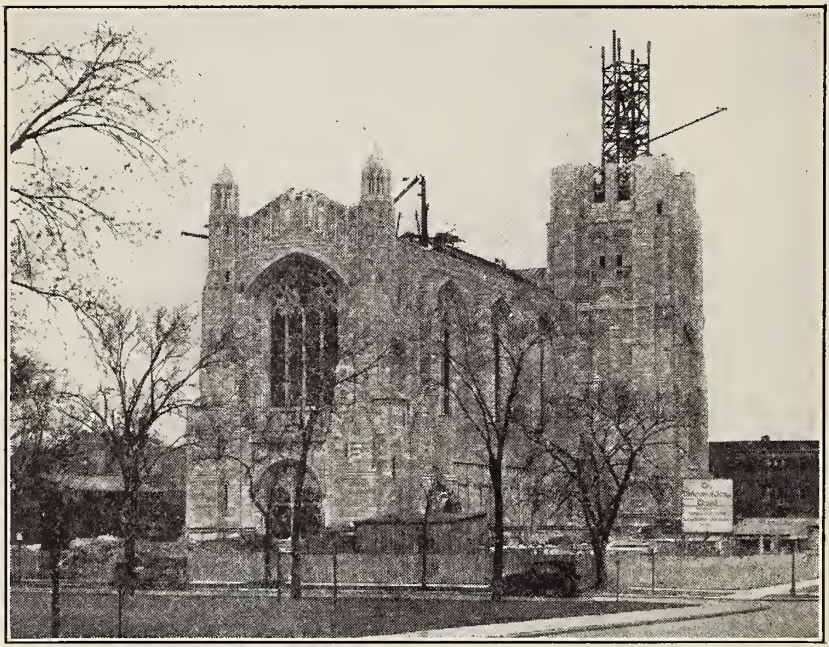


Fig. 112. The University of Chicago Chapel under construction

with as much energy as was used to raise it to the top. If the tower should be toppled over by some great force like an earthquake, as much energy would be developed in its fall as was used in its construction. The work done by its fall might not be of any service to man, the blocks of stone might merely smash each other or tear holes

in the ground where they struck, but their energy of position would be transformed so as to accomplish work even if was not useful.

The plants are building the simple things, already mentioned, into more complex bodies. The chemist believes that all substances are made up of tiny building units which he calls atoms; thus carbon dioxide is made of one atom of carbon and two of oxygen, water is made of two atoms of hydrogen and one of oxygen. The particle of water so built is too small to be seen and is called a molecule—there are billions of such molecules in a single drop of water. Now what the plant does is to build up very complicated molecules consisting frequently of hundreds of atoms. It takes energy to do this building just as it takes energy to build the tower. Then when we take these complex things into our bodies, we tear them down by the process of burning and so liberate for our use the sunlight which the plant absorbed and used in making the molecules. Not only that but we use this energy in part to construct still more complex molecules. Other animals do likewise, and we frequently use the flesh of other animals for food as well as the materials made by plants. Whether the food be animal food or plant food, however, the real source of its energy is the sun.

CHAPTER XI

SOME MODERN DISCOVERIES IN PROBLEMS OF NUTRITION AND GROWTH

It has long been known that certain diseased conditions in man are due to the lack of particular foods. Thus, sailors whose vessels were kept at sea by storms and contrary winds until their supply of fruits and fresh vegetables gave out, often came down with the scurvy. They became weak, sores broke out on their bodies, and many died of the disease. If, however, they could put into some island where they could find fruits, like lemons or oranges, or fresh green stuff to eat, their scruvy would disappear. This was particularly true in the days of the sailing vessels when ships were often at sea for months between ports.

In Hildebrand's "Life of Magellan" there is given a vivid account of the terrors of scurvy. The book is a thrilling tale of heroic adventures. Leaving Spain on the 20th of September, 1519, the little fleet of five vessels, the largest only 130 tons, sailed down the west coast of Africa, crossed the Atlantic to South America and fought their

way through winter storms, inhospitable coasts, and mutiny, to the southern tip of South America. It was on November 28, 1520, when the surviving three vessels, having passed the straits named in honor of Magellan, that the course was set northwest into the great Pacific.

The food was almost gone. The water was yellow, thick and stringy with unhealthy slime. They ate what they called biscuit; it was no more really than a disgusting powder, wriggling with worms. . . .

Then came the scurvy. The causes of this disease, . . . a general decreased vitality; cold, raw, and changeable weather; overcrowded quarters; hard work; anxiety and nervous depression; a deficient and monotonous diet, meat improperly prepared or partly decomposed, a lack of fruit and vegetables; bad drinking water.

They passed several desolate little islands.

The men were doing nothing more than enduring existence; . . . There was no food now. They cut up planks in small pieces, and ate the sawdust. They ate boots, and bits of leather from the armor, and straps from swords and shields. . . .

The scurvy was at its worst. The sick men's faces broke out in patches of brown and green, their ankles swelled to shapeless masses of pallid and pendulous skin, the fluid left the joints of their legs, so that they could not move. Their mouths were filled with bleeding ulcers, and the gums grew up in fleshy protuberances and covered their teeth. They screamed with pain. . . . On the sixth of March . . . land was sighted. . . . As they drew near the Islands took shape. . . . The ships ran into the bay and anchored . . . and the men went ashore. They found bananas and ate them, though they had never seen them before, and thought

them figs. They came upon large stores of cocoanuts, and gnawed ravenously as they went about the work of loading the boats . . . several loads of cocoanuts and bananas and sugar-cane and dried flying fish were taken aboard each ship. A pig was found, asleep in the dust, and they took it with them in great contentment.

Then they sailed.

But when a small island, obviously uninhabited, appeared to starboard, he steered for it and anchored in a quiet cove. An odor of spices came from the land, and the pleasant noise of rustling leaves; the sound of the surf on the windward shore was only a faint dull roar, like a background of monotony which made more keen the silence and the peace. This was the very place.

The high land was Samar, of the Philippines.

On the beach, two tents were built of sails and palm trunks, and the sick men, brought carefully ashore, stretched themselves gratefully on the sand in a cool and breezy shade. The rest explored the island and found good water. That night, the pig from the Ladrones was killed.

Every day the Captain-General went ashore to visit the sick; he knelt beside each man and gave him cocoanut milk to drink, and assured them all that their troubles were over, since they had now reached the countries of which he had told them. The men's health improved, with the rest and the fresh food, and, having their commander's personal attention, their courage came back to them; . . .[1]

Back in 1747 James Lind, a surgeon in the British Navy, proved in a series of experiments made on sailors that the medicines then in use for scurvy did no good but that the disease could be prevented and cured by the use of lemon and

[1] Hildebrand, A. S., "Magellan," Harcourt, Brace & Co., 1924.

orange juice. Early in the nineteenth century laws were passed making compulsory the supply of these juices to sailors on all British warships and merchant ships. Such measures and the introduction of steam vessels that reduced the length of time from port to port reduced scurvy so it became an almost unknown disease. It was ever ready to reappear, however.

It was only in the last decade of the nineteenth century that Frederick Gowland of Johns Hopkins University suggested that certain substances, now called vitamins, were necessary in the diet to maintain life. Since this time a large number of investigators, many of them Americans, have been working to discover what these substances are and in what foods they occur.

Many children suffer from a disease known as "rickets," in which the bones remain soft, the teeth do not form properly and the youngster suffers from digestive disturbances. Permanent deformities like bowlegs, knock knees, often result if the child does not die. Lunin suspected in 1881 that rickets is due to some substance lacking in the foods used. Cod-liver oil is commonly used now to prevent this trouble.

In countries like China and India, where rice is the chief and sometimes only food, there is prevalent a disease known as "beri-beri." If whole rice is eaten, the disease does not appear, but if the outer, brown coats of the rice are removed

and the polished grain only is used, the disease is likely to follow. Animals like chickens, fed on polished rice only, develop the disease. They lose weight, their muscles become weak so they cannot stand and they suffer from inflammation of the nerves.

So Eijkman, studying in the Dutch Indies in 1897, showed that beri-beri was due to some food deficiency. Later Grijns and others there showed that the outer covering of the rice, removed in polishing the white rice grains we customarily use, contain something that prevents the disease.

Several vitamins have been discovered, the most important of which are designated as Vitamins *A*, *B*, and *C*. Vitamin *A* is absent in the diet of rickety children, *B* is wanting where beri-beri develops and the absence of *C* is responsible for the appearance of scurvy. Vitamin *A* is found plentifully in fresh milk and spinach; *B* in most fresh vegetables and fruits and in seeds; *C* in tomatoes, lettuce, lemon and orange juice. These are by no means the only sources of these vitamins, merely sample foods in which they occur abundantly. Indeed the vitamins are so prevalent in common foods that one who lives on a mixed and varied diet will probably eat enough of each to meet his needs.

The quantity of vitamin required is very little. The vitamins are not used up as tissue builders or as sources of energy but merely by their pres-

ence aid in the control of essential life activities. That their absence produces disease and ultimately death shows how important they are. Some of them have been separated from the substances in which they occur and appear as whitish powdery materials.

There is another group of substances in the body, called "hormones," that are of vast importance although they, too, act in very tiny amounts. These are secreted by various organs and sent as chemical messengers through the blood stream to regulate activities going on often far from the source of the hormone. It was formerly thought that the only way one organ could influence another was through the nervous system, but now it is realized that many, if not most organs, are sending out these chemical messengers to influence the activities of other organs.

Pause to consider how terribly complex this body of ours is. It is made up of so many workers, blood cells, muscle cells, gland cells, nerve cells, that one could not count them all if he could live several thousand years—we can only roughly estimate their number. These are grouped together into tissues and the tissues into organs. Each organ, each tissue, each microscopic cell has its particular task to do and it must do that in harmony with all the others, always at the right time, never doing too much or too little.

For instance, you start to run, making the

muscles of legs, arms, trunk all work harder and more rapidly than they were doing a minute before. That means that more power must be generated so the sugar must be brought to them more rapidly to be burned to supply the added energy and more oxygen must be brought to them, else the more rapid burning or oxidation can not go on. The liver must pour out its reserves of sugar into the blood, else one would die in convulsions, so sensitive are certain nerve centers to a reduction in the sugar content of the blood. The heart must pump more vigorously to drive the blood more rapidly. Breathing must be deeper to take in the extra oxygen. The carrying capacity of the red corpuscles in the blood must be increased. The waste materials that result in larger quantities from the breakdown of the tissues must be carried off and excreted faster. Such an apparently simple act as starting to run calls for hundreds of adjustments in the body, brings into play the coöperation of countless working cells from head to heels.

Certain cells in the pancreas, in the example above, must discharge into the blood larger doses of one of these hormones known as "insulin," for whenever oxidation of carbohydrates such as sugar is going on in the body this insulin is necessary to the regulation of the process. It was discovered in 1889 that removal of the pancreas in animals led to defects in handling sugar in the body that

finally caused death. It was Schafer who, in 1916, suggested that the pancreas produced a hormone essential in the process of carbohydrate metabolism. There is a disease in man known as "diabetes" in which the carbohydrates are not properly handled by the body. It was formerly always fatal. But a number of workers led by F. G. Banting (Fig. 113) of the University of Toronto obtained the insulin from the pancreas of animals so it could be given to patients suffering from the disease and so prevent death. Evidently the pancreatic glands of such patients were not producing insulin in sufficient quantities to take care of the production of muscular energy by the oxidation of carbohydrates.

Fig. 113. Dr. F. G. Banting

There are a number of glands in the body that seem to have as their sole function the production of such hormones. Such have no ducts by which they pour out their secretions into various organs, as the salivary glands pour saliva, for instance, into the mouth. But these ductless or endocrinal

glands pour their secretions containing the hormones directly into the blood that runs through them. In addition, many of the glands with ducts, and many other organs not primarily glandular, secrete hormones which they discharge into the blood to aid in these regulatory processes.

Among the most important of the endocrinal glands are the pituitary, a stalked gland hanging from the under side of the brain and contained in a bony cup of the skull, the thyroid and parathyroids in the neck near the Adam's Apple, the adrenals that lie one on top of each kidney. In the latter part of the eighteenth century and all through the nineteenth century a variety of diseases were shown to be due to increased or decreased activity of these glands. The removal of any of these glands from such animals as dogs was shown to result in death. In some way they evidently were essential to life. It has remained for experimenters in the twentieth century to obtain from them the pure or nearly pure hormones which they manufacture and to determine some of the important effects they have on the bodily functions.

The hormone or hormones of the front portion of the pituitary stimulate growth and if persistently administered produce giants. Those of the rest of the pituitary, when injected into the blood, raise the blood pressure and do this in very dilute doses. The hormones of the thyroid seem to have

to do with the fundamental activities of growth and of energy production by oxidation. Lack of the hormone produces abnormal development and, more or less, idiocy. Too much of it stimulates heart action and produces muscular tremors and weakness. The hormones of the adrenals (or suprarenals) affect the sympathetic nervous system, stimulating it in much the same way as does an electric current.

No more wonderful chapter in the history of discovery relating to the human body has ever been written than the one begun a century ago and now hastening toward completion, dealing with the endocrinal secretions and the other hormones of the organism. To grant, as insulin does, a new lease of life to the sufferer from diabetes otherwise condemned to speedy death, to restore the idiotic child, the cretin, to normalcy with thyroxin, to control growth with pituitary extract and produce giants at will, to keep alive and growing for years a bit of tissue removed from an embryonic chick and kept entirely apart from a living organism, as Carrel has done with a growth hormone, are performances that border on the miraculous. They are prophetic of still more marvelous accomplishments when the many other hormones shall have been isolated and their effects experimentally determined.

We have seen in this book how the discoveries

of the early scientists furnished the foundations on which other discoveries were built. So each generation has added to the accumulating knowledge that banishes ignorance and fear and that makes for healthier and therefore happier living. There can be no ending to such a history as this for it is still in the making. It is a glorious story, this story of these men who through toil, self-sacrifice, and sometimes suffering or even death, have made the world a safer and more comfortable place to live in because disease is less prevalent and because we know better how to keep well. Each discovery has, however, made more apparent the need for other discoveries. The more we know, the more we see there is to know. It would be a sad ending for a book like this to leave the impression that the great discoveries regarding the physiological activities of the human body and man's control of them have largely been made. The greatest undoubtedly yet await a discoverer. We revere the men whose work has been described, but we envy youth its opportunities to uncover still more marvelous mysteries in aid of man's health and welfare.

APPENDIX

DEMONSTRATION I (P. 7)

THE tiny creatures that are being discussed in the text are probably entirely unfamiliar to the average pupil. It is quite necessary, therefore, that he should see some of these forms to make the terms used meaningful. A culture may be prepared in which there will appear a succession of these tiny living creatures. Put a handful of hay or of dry stems and leaves of weeds into a quart fruit jar, or, better still, a battery jar. Cover the hay with water and let it stand, preferably in a warm, but not hot place; loosely cover the top of the jar. The organic material will soak out into the water, forming a dilute soup in which tiny organisms can live. Some of the dried-up organisms or their spores will be sticking to the hay, so that, within a few days, living creatures will appear. A scum will be seen on the surface of the water, and probably the preparation will have a somewhat disagreeable odor. This scum will be found to consist largely of very tiny rod-shaped bacteria. In the course of four or five days there will probably appear much larger corkscrew-shaped forms closely related to bacteria (Fig. 114). Within ten days or two weeks, flagellates and ciliates will probably appear. The latter will be of small species at first, and in all likelihood, these will be followed by the larger "slipper animalcules," paramœcium, Fig. 4.

If you tie a piece of string to a chunk of raw liver as large as your thumb, and hang this below the surface of the water

in the hay infusion, it will increase the number and variety of the forms that will appear in the jar.

Of course these can be seen only under a microscope and a fairly high power will be required for the bacteria. Pick up a bit of the scum with a medicine dropper at the same time drawing in two or three drops of water. Put a drop of the water with some of the scum on a microscope slide, cover it with a clean cover-glass and absorb the excess of water with filter paper or blotting paper. This drop may be examined then under the microscope, using first the low power to see if living things are present. They will appear as tiny rapidly-moving specks. If the first drop examined does not contain living forms, try another. When they are found in numbers turn on the high power. The paramœcia will be found on the side of the jar near the light, if it is standing on the windowsill, so take the drop of water from the surface at the edge of the jar nearest the window.

Fig. 114. Corkscrew-shaped form closely related to the bacteria

If possible, use a projection microscope to throw these forms on the screen. The slide prepared as above is put on the stage of the projection microscope just as it would be on the stage of an ordinary microscope. A cooling tank or other device must be used to take the heat out of the light before it strikes the drop of water; otherwise these tiny organisms will be cooked. A little experience will give the teacher skill in handling the projection microscope so that these living creatures will appear on the screen quite as plainly as under the ordinary microscope. The teacher is very sure then that all members of the class are seeing the

desired objects. One is never quite certain that children using a microscope see the thing they are looking for.

A preparation of the scrapings from the teeth to which has been added a little water will enable the teacher to demonstrate the bacteria from this source as Leeowenhoek did.

DEMONSTRATION 2 (P. 9)

REDI'S experiment is easily repeated. Put a piece of meat as large as the palm of the hand into one fruit jar and tie a piece of cheesecloth over the mouth of this jar. Put a similar piece into a second jar and leave uncovered. Place these two jars on the windowsill outside the window of the schoolroom where the pupils can watch what goes on in them. This of course must be done early in the Fall before the flies have gone into winter quarters or else in the Spring when flies are about. It will be interesting to keep a record of the date when the eggs are deposited, when the maggots are first visible, when the pupæ appear and when the adult flies hatch out.

DEMONSTRATION 3 (P. 23)

THE process of fermentation is readily demonstrated. Dissolve two tablespoonfuls of ordinary sugar in a half pint of water. Add to this a piece of compressed yeast as large as the end of the little finger, first breaking it up into small bits. Put the sugar solution with the yeast in it into a bottle, and plug the neck of the bottle with absorbent cotton. Prepare a similar solution of sugar and put it into a pint fruit jar; leave this uncovered. The first solution of sugar will ferment, due to the action of the yeast; probably some micro-organisms in the dust of the air will settle in the second solution in the pint fruit jar and start fermentation. When the fermentation is under way, bubbles of carbon-dioxide

gas will rise in the sugar solution and the progress of the fermentation can be detected by the odor. There will first be a smell of alcohol and later an acid odor.

Stir up a tiny bit of the yeast in a few drops of the sugar solution; put a drop of this on a microscope slide and examine it under the microscope, or, better, project the yeast plants onto the screen. If vaseline is smeared part way around the edge of the cover-glass so the water will not evaporate, the preparation may be left on the stage of the microscope overnight so that the class can observe the same yeast plants the next day. If a drawing is made of certain ones when examined the first day and a second drawing is made the next day of the same plants, it will probably be seen that some of them are budding, and in place of the single cells there will be chains of two or three cells.

DEMONSTRATION 4 (P. 26)

TRY darkening the schoolroom, or if that is impossible, some other room on a side of the building that is exposed to sunlight. Admit a small beam of light into the room through a hole in the covering of one of the windows. Note the dust-motes that are visible in the beam of light. Of course, each one is familiar with the fact that such dust particles settle on the furniture so that a room needs frequent dusting or if it is not so dusted, one can readily see the accumulation of dust on tables and other objects.

DEMONSTRATION 5 (P. 26)

PASTEUR'S experiment may readily be repeated. Either make some pea soup or get a can of it from the store. Strain it so as to remove solid particles. Fit the mouth of a glass flask with a stopper that has a hole in it just large enough to receive a piece of glass tubing. Insert the glass tubing into the stopper so it just goes through the cork; bend this

glass tubing into the shape of the letter "s." Fill the flask half full of the soup and put in the stopper with its tube. Bring the soup to a boil. Then let it stand. Put the rest of the soup, only part of which was put into the flask, into a pint fruit jar and let it stand uncovered. In the course of a few days, compare the two samples of soup. The uncovered soup will probably be fermenting as can be told from the disagreeable odor. The soup in the flask will probably not ferment. When the two samples of soup are examined under the microscope, the micro-organisms will probably be plentiful in the soup in the pint jar, but absent from the soup in the flask.

DEMONSTRATION 6 (P. 30)

THE life history of the silkworm may readily be demonstrated to the class. This should be done either in the early Fall or the late Spring when the leaves of the common mulberry can be obtained as food for the larvæ. Silkworm eggs can be had from T. A. Kelleher, P. O. Box 82, Washington, D. C., or from other dealers (p. 298). For twenty-five cents, these houses will send a batch of the silkworm eggs, attached to a bit of paper. There are usually several hundred eggs in the lot. These eggs appear like very tiny pearls. Kept at ordinary room temperatures, they will probably begin to hatch within a week or ten days from the time they are received. As soon as they begin to hatch, lay the paper with the eggs upon it on some fresh mulberry leaves in a pasteboard or wooden box. The larvæ will promptly crawl onto the leaves and begin to feed. It is not necessary to cover the box for the larvæ will not leave their food supply. Fresh leaves should be supplied every two or three days when the larvæ are small, every day as they grow large and eat the leaves rapidly. When the larvæ have grown to the size of the little finger, they will

begin to spin their cocoons. These can be kept until the moths hatch out and after they have hatched and mated, the females will lay their eggs.

DEMONSTRATION 7 (P. 45)

THE method of making the hanging drop culture is easily demonstrated. Secure a glass slide on one side of which is a hollow, ground out of the glass. Such slides can be obtained from any dealer in microscope materials. In the center of a clean cover-glass, put a small drop of any fluid culture material, like sterile beef broth. Dip a needle point into the scum in the jar of hay infusion and wash it off in the drop on the cover-glass. Smear a little vaseline onto two or three spots around the edge of the hollow of the glass slide. Then turn the cover-glass upside down and lay it on the slide so that the drop will hang in the hollow. This preparation can be examined under the microscope day after day, when it will probably be seen that the organisms present are increasing in numbers.

DEMONSTRATION 8 (P. 46)

PREPARATIONS of many of the bacteria that are to be discussed in this book can be obtained on permanent microscope slides from any of the reliable dealers like the General Biological Supply House, 69th Place, near Cottage Grove Avenue, Chicago; The Kny-Scheerer Company, New York City; Ward's Natural Science Establishment, Rochester, N. Y.; or similar concerns. It would be advisable to show a few of these to pupils so they will have a clear notion of some of these bacteria, enough to realize that they have characteristic shape, size or staining properties that distinguish one from another. The bacteria of anthrax, of tuberculosis, of diphtheria, of pneumonia and some of the protozoan parasites like the one that causes malaria, or the

trypanasomes of sleeping sickness, might be demonstrated as needed.

DEMONSTRATION 9 (P. 49)

TAKE the pupils out to some area near the school where they can see the mounds of castings about the holes of earthworms. An earthworm or two might be kept in the schoolroom in a large flower-pot full of rich soil. Cover this with a piece of glass, so the moisture will not evaporate too readily. Bits of decaying leaf or other partially decomposed organic material can be left on the surface of the ground, and possibly the pupils may see the earthworms feeding. If the glass over the pot is covered with a piece of cardboard so that the interior is dark, the worms will stick their mouth-ends out to feed even in the daytime.

DEMONSTRATION 10 (P. 53)

IF a little fresh blood can be obtained from a butcher shop or from some small animal, the clot will separate from the plasma if allowed to stand for a short time. If a little of the remaining serum is put into a test-tube and gently heated, it will jell.

Test-tubes with culture media already prepared in them, and sterilized, are easily obtained now from the supply houses already referred to. Obtain half a dozen such tubes. Keep the mouths of the tubes closed with absorbent cotton except as directed below. Fasten an ordinary sewing needle into a wooden handle, forcing the eye of the needle into the soft wood. Heat the tip of the needle in a flame so as to kill any bacteria upon it. Scrape out a bit of food material with this tip from between the teeth. Remove the cotton plug from one of the test-tubes and quickly stab the needle into the culture medium. Close the tube again with the wad of cotton. Try planting in a similar way bacteria from other sources. Allow these inoculated tubes to stand for

several days. The bacteria will multiply and the colonies appear more promptly if the tubes can be kept in a moderately warm place. They may be stood, for instance, in a small box in which an electric light is left burning. It would add to the interest of the apparatus if a thermometer could be put into the box so as to watch the temperature. Of course, this is a very crude contrivance compared with the apparatus the scientists actually use, for the temperature and other factors must be regulated with great exactness.

It may be that two or three kinds of bacteria were introduced on the needle tip, or possibly only one kind. If different sorts of bacteria were planted, when they multiply they will likely produce different kinds of colonies. These colonies will be distinguished by their form or their color or their effects on the culture medium. Figure 115 shows some of these cultures with different types of colonies in them.

If some strikingly peculiar colony does appear in one of the culture tubes, try transplanting a bit of it into another one of the tubes to see if, in the second tube there will grow the same sort of bacteria recognized by the peculiarity of the culture. This latter part of the experiment is intended to make clear to pupils the way in which the scientist inoculates a culture tube from a previous culture. It also shows that even among these minute organisms like produces like.

DEMONSTRATION II (P. 58)

THE use of various chemical agents, usually called germicides, in killing living things may readily be demonstrated. Put a drop of the water from the battery jar, prepared in Demonstration 1, on a glass slide. In this there will probably be swimming about many tiny living creatures and probably there will also be abundant bacteria. Cover this

drop with a cover-glass, absorbing the excess of water with filter paper. Put this preparation on the stage of the microscope, the projection microscope preferably. Now add a tiny drop of carbolic acid, of creolin, or of any other common germicide, at the edge of the cover-glass. This will gradually diffuse into the water under the cover-glass. If creolin is used, one can readily follow its diffusion because

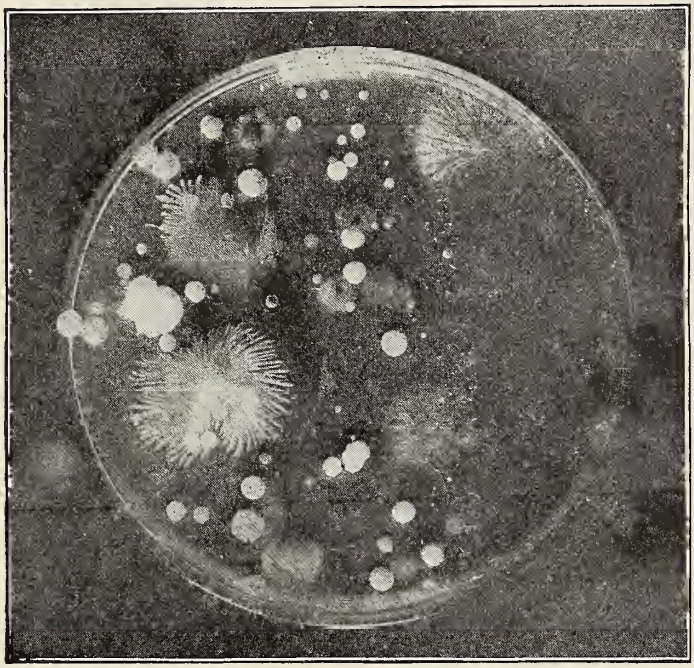


Fig. 115. Bacterial colonies on gelatin in petri dish

of its dark color. Get the boundary of this diffusion area under the microscope and it will be seen that as the living creatures come in contact with the germicide in the water, they promptly die.

One can vary this experiment by putting a tablespoonful of the water from the hay infusion into a test-tube, then adding a drop or two of the germicide. The living organisms will die and settle to the bottom of the test-tube in the

course of a few minutes; then a bit of this material can be picked up with the medicine dropper and examined under the microscope to show that the organisms have all ceased moving and have been killed by the germicide.

DEMONSTRATION 12 (P. 83)

BLOOD corpuscles are easily shown under the microscope. Heat the tip of the needle, which for a former demonstration was imbedded in a soft wooden handle. Press the thumb of the left hand firmly against the basal portion of the last joint of the third finger. This will force the blood to the end of the finger and, at the same time, help to deaden sensation. Prick the end of the finger with the needle so as to draw a drop of blood. Put this on a clean glass slide and promptly draw the edge of a second glass slide, held at an angle of about 45 degrees, through the blood droplet so as to spread the latter in a thin film on the glass. The blood smear dries promptly and may now be examined under the microscope without a cover-glass. One will readily see the blood corpuscles, the red ones circular in outline, the white ones rather irregular. Only an occasional white corpuscle will be seen among the red corpuscles as they are much less numerous than the red.

If, instead of smearing the blood on the glass slide, the little drop of blood is put into a drop of normal salt solution, and this is covered with a cover-glass, some of the red corpuscles will be seen edgewise when the preparation is examined under the microscope. A salt solution that will serve the purpose may be made by adding a single grain of salt to a drop of water.

DEMONSTRATION 13 (P. 83)

IT is more difficult to show the students the movements of the white blood corpuscles than it is to show the movements

of the amœbæ, a tiny animal consisting, like the white blood corpuscles, of just a speck of living substance. With a medicine dropper, gently scrape the surface of the piece of liver that was put into the hay infusion, at the same time drawing up a few drops of water into the pipette. Put a drop of this on a glass slide, cover with a cover-glass and examine for amœbæ. Usually, in the hay infusion, small amœbæ appear feeding on the liver within a month of the preparation of the infusion. Larger species appear within a couple of additional weeks. These amœbæ are quite transparent; therefore, when looking for them under the microscope, use rather dim light. They can be recognized by the flowing movement. A part of the animal is thrust out and the rest of the animal flows into this portion. The white corpuscles have this same sort of amœboid movement.

DEMONSTRATION 14 (P. 112)

Mosquito larvæ can usually be found in rain barrels or other stagnant water, either in the early Fall or in the Spring or Summer. They are common in shallow ponds or in the quiet water along the margins of a stream. A wire hoop can be fastened to the end of a broomstick and a shallow cheesecloth net fastened to the hoop. With this, one can sweep through the water and obtain these mosquito larvæ or wrigglers. They can be kept in the schoolroom in tumblers or pint fruit jars half full of water, and their actions can then be observed. Note how they hang head down from the surface film of the water when they are at rest. When disturbed, they swim down into the water, but return to the resting condition with the air tubes thrust slightly out of the water so they can breathe.

Drop one or two drops of kerosene oil on the surface of the water in one of the tumblers where there are several of the mosquito larvæ. See how promptly the larvæ die, when they are unable to get to the air.

Put a small fish in one of the pint fruit jars where there are several of the mosquito larvæ. One can obtain from the pond when fishing for the larvæ, some small fish like the top minnow, the little stickleback, or the johnny-darter. These fish are usually found swimming near the bottom so that the net will need to be moved close to the bottom. Carefully poke the rim of the net into the mud to stir up the animals that may be lying in or on it. Watch how soon the fish will eat the mosquito wrigglers. When the experiment is finished, put the fish in the school aquarium or return them to the pond or stream.

DEMONSTRATION 15 (P. 138)

SHOW under the microscope a preparation of the bacteria of typhoid fever. Prepared slides can be obtained from the firms whose addresses have already been given (*see* Demonstration 8).

DEMONSTRATION 16 (P. 142)

SHOW under the microscope some one of the trypanosomes. Prepared slides of these animals can be obtained from the firms whose addresses have already been given.

DEMONSTRATION 17 (P. 148)

SHOW under the microscope the hookworm. Prepared slides can be obtained as indicated in preceding demonstrations. While the figures of these animals show reasonably well how they appear, it is a satisfaction to the pupil ordinarily to see the real object.

DEMONSTRATION 18 (P. 225)

LAY the right hand on the upper left arm about at the elbow and then move it down toward the wrist while pressing

firmly on the arm. The veins of the forearm will stand out prominently and swellings will appear upon them like knots in a cord. These swellings mark the position of the valves of the vein. Why do they not appear when the hand is moved from the wrist toward the elbow?

DEMONSTRATION 19 (P. 227)

It is worth while to give pupils a demonstration of the main structures of the heart. It would be well to precede this by a study of a manikin. Show the students the main body cavities, the dorsal cavity containing the brain and spinal chord, the ventral cavity divided into the thorax or chest and the abdomen. Note in the thorax the position of the lungs and heart, the trachea and its connection with the bronchi, the main arteries and veins connected with the heart. Note the diaphragm separating the thorax and abdomen. See in the abdomen the stomach, small and large intestine, liver and gall bladder, pancreatic duct, bile duct, the kidneys, and the bladder.

If the school does not possess a manikin, there is a fairly satisfactory paper manikin in *Nelson's Encyclopedia* in the article "Anatomy."

A beef heart can be obtained from the butcher. If he is informed what the heart is wanted for, he will leave it enclosed in the pericardium with the large blood vessels attached. Let the class see it in this condition, then slit the pericardium so the heart can be seen clearly. Note the auricles and ventricles. Make a longitudinal incision down one side of the heart so as to enable the pupils to see the inside of the auricle and ventricle and the valves between them. Notice tendonous chords that hold the flaps of the valves so they cannot swing back too far. Note also the muscles to which the lower end of these chords are attached. These muscles pull on the chords just enough to hold the

edges of the valve flaps together. Run a glass rod or small stick into the open ends of the blood vessels so that the end of the stick will appear in the cavity of the heart. The veins open into the auricles, the arteries out of the ventricles (Fig. 94).

DEMONSTRATION 20 (P. 229)

THE flow of the blood in the capillaries may readily be demonstrated to the class. Obtain a small goldfish, preferably one with tail pretty largely free from color. Put a level teaspoonful of crystals of chloretone into 4 ounces of water. Shake it up until all is dissolved that will go into solution. This solution keeps well and may be used year after year until exhausted. Put the goldfish into a small beaker or a tumbler with just enough water to cover it. Add to the water a teaspoonful of the chloretone solution. Chloretone has the same effect upon the fish that chloroform has upon animals with lungs. The goldfish will begin shortly to turn over on its side. If it does not do so in the course of three or four minutes, add a little more of the chloretone solution. When it does begin to roll over on its side, take it out of the water. If it flops pretty vigorously, let it remain a while longer until it is fairly quiet on removal. Lay the goldfish on an ordinary microscope slide and cover it, except its tail, with a couple of layers of moist filter paper or a wet cloth. Spread the tail out and examine it under the microscope. Keep the tail moist, but do not cover with a cover-glass. Small arteries, running for the most part along the fin rays, will be seen carrying the blood out toward the end of the tail, and small veins carrying it back toward the body. Between these larger vessels will be seen the tiny capillaries through which the corpuscles go in single file. This demonstration may be conducted with the projection microscope so that the entire class may see this on the screen as described. After the pupils have seen the

flow of the blood under the low power of the microscope, the high power lens may be used and the corpuscles will be seen with much greater detail. Return the fish to the aquarium or to a jar into which water is running. The animal will recover shortly none the worse for the experiment.

DEMONSTRATION 21 (P. 239)

PREPARED dissections can be had from the biological supply houses showing the organs of a frog, including the distended lungs with their blood vessels injected. If such preparations are not available, obtain the lungs of a calf, pig or sheep from the butcher. Instruct him to leave the windpipe attached. Insert into the open end of the windpipe, or trachea, a glass tube or one made of a roll of stiff paper. Tie this in. Then blow through it so as to inflate the lungs. Allow them to collapse again, thus showing their elasticity.

DEMONSTRATION 22 (P. 247)

HOLD the upper part of your extended right arm firmly with your left hand. Now bend the elbow so the clenched right fist is brought up to the shoulder. Feel the biceps muscle swell as the ends come nearer together, to bring up the fore-arm. See the accompanying diagram for the points of insertion and attachment of this muscle to the bones of the skeleton (Fig. 116).

DEMONSTRATION 23 (P. 252)

THE distinction between a physical change and a chemical change may not be clear at this point to the pupils. Obtain a couple of six-inch lengths of magnesium ribbon or wire. With fine sand-paper, or a knife blade, scour off the white deposit so as to expose the metallic magnesium of one strip. Tear off several small bits from this strip. These are types of physical change.

Hold the other strip of magnesium ribbon with a pair of forceps and light one end of it with a match. Hold the burning strip over a piece of paper so as to catch the resultant product. Compare this product with the magnesium ribbon. Note that while the latter is a metal and is tough and elastic the new product has no metallic luster, is a soft powder with a greasy feel. A chemical change has been produced

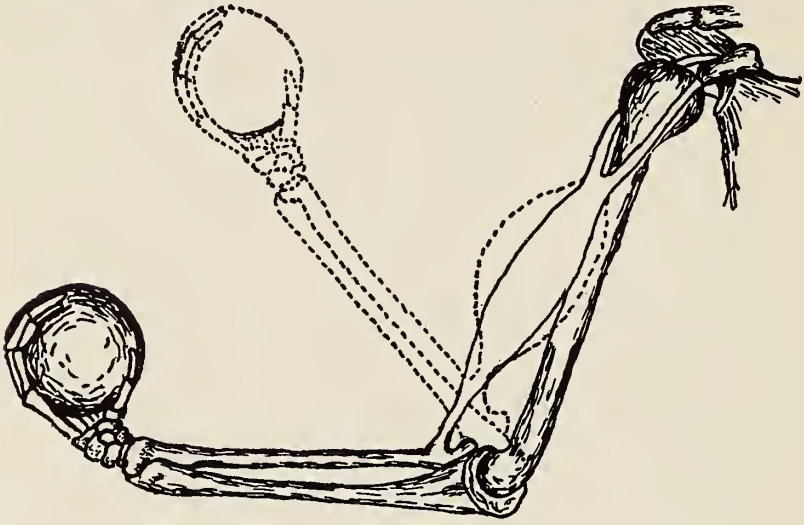


Fig. 116. Biceps muscle

and the properties of the new substance are totally unlike those of the original.

DEMONSTRATION 24 (P. 252)

PUPILS may not be familiar with the difference between alkaline, acid and neutral states. Make up a solution of phenolptaleine in distilled water. Filter it so it will be clear. Add to a test-tube full of this solution a few drops of ordinary ammonia. The colorless solution becomes bright pink. If now a few drops of acid like that of vinegar be added, the pink solution becomes colorless again. One may use strips of litmus paper which comes both in the blue

and the red in place of the phenolphthalein solution. The red litmus changes to blue in an alkaline solution like ammonia, the blue litmus changes red in an acid solution like vinegar. In a neutral solution neither acid nor alkali, such as ordinary water, there is no change in color in the litmus paper. Litmus and phenolphthalein are used as indicators of the acid, alkali and neutral conditions.

DEMONSTRATION 25 (P. 256)

OBTAIN about a teaspoonful of pepsin at the drug store. Dissolve half of it in water. Into one test-tube or a small bottle put a few bits of shredded meat, either raw or cooked. Fill the test-tube half full of water and cork it with a wad of absorbent cotton. In another test-tube or small bottle put a few similar shreds of meat and fill the test-tube half full of the pepsin solution. Cork it with absorbent cotton. Prepare another test-tube in the same way, only add a drop or two of dilute hydrochloric acid. Let these all stand in a warm room for a couple of days. In which does the meat most completely disappear as it goes into solution after digestion?

The remainder of the pepsin may be used in similar experiments with other materials like white of hard boiled egg, cheese, or the gluten of flour. If flour is mixed with water, making a fairly stiff dough, and if this is then washed in a stream of water as it is rolled or kneaded between the fingers, the starch will be largely washed away and the gluten left.

DEMONSTRATION 26 (P. 264)

MAKE a wad of writing paper small enough to go readily into the mouth of a tumbler. Float it on the surface of the water in a shallow pan. Light it, and when it is burning, set the empty tumbler mouth down over the burning paper so the mouth of the tumbler is under the water. Notice

that the water rises in the tumbler as the paper burns. Was the tumbler really empty in the beginning? The carbon dioxide formed as the paper burns dissolves in the water promptly.

DEMONSTRATION 27 (P. 266)

PUPILS are probably not familiar with the appearance or properties of oxygen. It seems desirable, therefore, to show them the gas and some of its characteristic types of behavior. Fill a rather large test-tube one-third full of a mixture of equal parts of manganese dioxide and potassium chlorate, the former in powdered form, the latter either powdered or

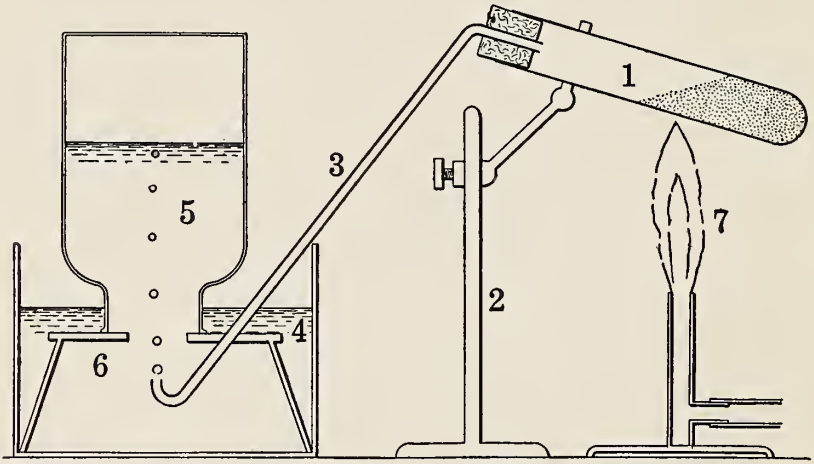


Fig. 117. Apparatus for generating oxygen
1, Test tube; 2, support; 3, delivery tube; 4, water in pneumatic trough; 5, inverted bottle; 6, shelf; 7, flame

in crystals. Take a cork that will fit the mouth of the test-tube and in this cork make a hole. Fit into this hole a piece of glass tubing and bend it enough so as to form a delivery tube. (See sketch of apparatus in Fig. 117.) The delivery tube should project slightly into the test-tube. Hang or clamp the test-tube with its delivery tube on a support.

Fill a pneumatic trough with water so the level of the water stands just above the shelf. Fill three four-ounce wide-mouth bottles with water. To do this set each in succession in the pneumatic trough and turn it upside down, keeping the mouth below water. If the pneumatic trough is not deep enough to permit this, cover the mouth of the bottle full of water with the palm of the hand or with a small piece of glass. Invert the bottle. Put its mouth under water and withdraw the hand or the glass. Set the bottle, mouth down, on the shelf of the pneumatic trough. Slide one of the bottles over the hole in the pneumatic trough and put the open end of the delivery tube from the test-tube under the hole. If a pneumatic trough is not available, a deep pan can be used and the bottle can be supported between two other bottles or other objects standing in the water.

Heat the mixture in the test-tube with the flame of a Bunsen burner or an alcohol lamp. Move the flame about a bit, so the mixture will be heated throughout, not merely at one spot. The heat will cause the air in the test-tube to expand and a few bubbles will flow out into the bottle. Very soon, however, oxygen gas will be generated and will bubble out into the bottle. When the first bottle is filled, slide it to one side of the shelf of the trough and bring the second bottle over the mouth of the delivery tube. Thus collect three or four bottles of oxygen. Lift the delivery tube out of the water so that as the test-tube cools the water will not flow back into it.

Slip a small square of glass or even a stiff cardboard under the mouth of the bottle. Lift it off the shelf and set it bottom down on the table, the mouth still covered with the glass.

It will be noted that the gas in the bottles is colorless, as it is, of course, in the air. Light the tip of a splinter of wood and when it has blazed for a moment, blow out the flame, leaving a glowing ember on the end of the splinter. Slide the glass cover on the mouth of one of the bottles to one side

and introduce this splinter into the oxygen. The glowing ember at once bursts into flame. It may be withdrawn and the experiment repeated a couple of times before the oxygen is used up. Take a piece of iron picture-wire 6 or 8 inches long. Unravel the wire at one end and twist into the wire a bit of wood. Light this and when it is burning well, introduce it into a bottle of oxygen, the bottom of which is covered with water. Not only will the wood burn, but the wire also will burn, giving off sparks. If a small animal like a grasshopper is introduced into one of the bottles of oxygen, it will live a very fast life for a few moments. In a larger supply of oxygen it might wear itself out and die of exhaustion. In the small supply it will be very active for a short time but will probably use the oxygen up before it exhausts itself completely.

DEMONSTRATION 28 (P. 267)

WITH a finger of the left hand hold the lower lip down so that it is turned inside out. Lightly scrape the inner surface of the lip with the blade of a scalpel or penknife that has been held for a moment in a flame to kill any germs upon it. Wash off the scrapings into a drop of water on a clean microscope slide. Add a tiny drop of neutral red stain. Cover with a cover-glass and examine under the microscope. This will readily show body cells. Similarly, show cells in some sections of plant and animal tissues. Sections of the growing root tip of an onion or other bulb; sections of spinal chord of glands, and of unstripped muscle tissue are good for this purpose.

DEMONSTRATION 29 (P. 195)

THERE is given, Fig. 118, a graphic demonstration of the increase or decrease of the death rates from certain important diseases in the United States. Note that some of them,

like the death rate from tuberculosis, drop considerably in the ten-year period. The same is true of diarrhea, which affects little children largely. On the other hand, heart disease, most serious among adults, has been increasingly fatal. The epidemic character of influenza is apparent emphatically in 1918.

Attach to the edge of the sheet on which this graph is printed a sheet of unruled paper and rule lines for 1925, 1926, etc. Extend the lines for these ten diseases for these years. The data needed, the death rates per 100,000 of population, can be found in the volume of Mortality Statistics published by the Bureau of Census, Washington, D. C. You will likely find a late volume in your local public library or the librarian will obtain the latest copy for you.

Add lines for typhoid fever, measles, whooping cough, diphtheria and similarly extend the lines on the basis of the data you secure for the later years. Here are given the death rates per 100,000 population for the four diseases just mentioned during 1915–1924.

	1915	1916	1917	1918	1919	1920	1921	1922	1923	1924
Typhoid fever	10.4	10.2	9.1	8.4	5.7	'5.3	6.0	4.4	4.2	3.7
Measles	5.6	11.7	9.3	9.6	3.6	10.2	4.1	6.1	10.1	5.7
Whooping cough	7.9	9.8	8.8	14.7	4.7	12.6	8.5	5.7	8.7	6.5
Diphtheria	14.5	13.9	16.5	14.7	15.9	15.9	17.7	14.4	12.9	10.4

Is the death rate from such diseases as are prevalent in childhood decreasing or increasing? What effect would this have on the death rate from those diseases which are most common among adults?

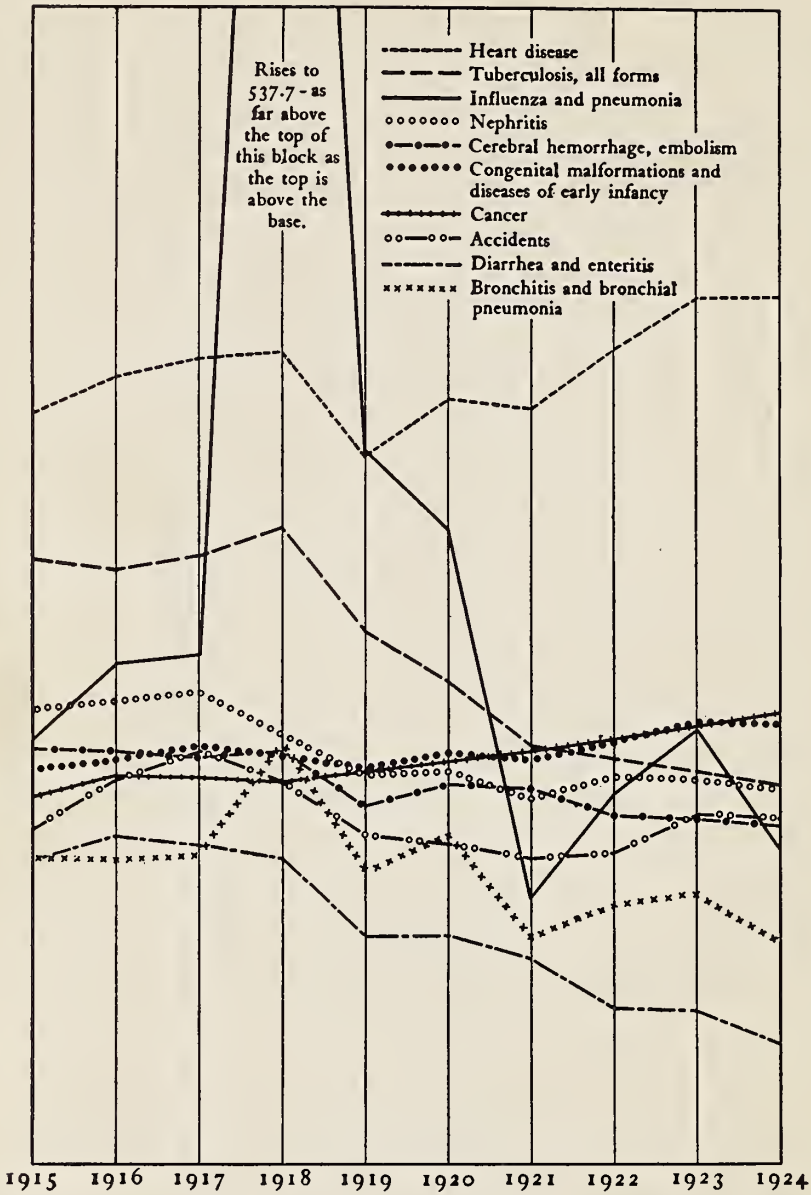


Fig. 118. Death rates for the ten most important causes; 1 mm. above the base line equals 2 per 100,000

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